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DETACHMENT OF THE RETINA

TREATMENT BY MEANS OF THE PYROMETRIC ELECTRODE

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In the operative treatment of detachment of the retina one tries to produce an adhesion between the retina and the choroid close to the place surrounding the rent. The most logical method to produce this result is scleral diathermocoagulation or surface coagulation.

In contrast to all of the others, this method provokes choroidal irritation without perforating the sclera, so that the eye keeps its tonus during the whole operation. Only when the operation is finished are the evacuating punctures begun, and as these are done in a cauterized area, all risk of hemorrhage is avoided.

The diathermic reaction lasts about ten days and produces a zone of solid adhesion between the retina and choroid.

The features of this electrocoagulation are well known, except for the most important feature, the exact measurement of diathermic effect, which has failed up to the present.

The diathermic effect has been measured by some ophthalmologists by the electric intensity. For instance, they say, "It is coagulation of 250 ma.," but this is far from accurate. D'Arsonval has shown that diathermic coagulation does not depend on the electric intensity alone. In my studies I found that an intensity of 250 ma. produces sometimes considerable and sometimes only slight effect. Other operators depend on the brown discoloration of the sclera, or on the "parchment transformation," but these changes are really too subjective.

It thus became evident that scleral coagulation requires an objective method of measurement, which will give constant results whatever the conditions may be. This can be attained by measuring the temperature of the coagulated area, which is now possible by means of my pyrometric electrode. The pyrometric electrode is a diathermic electrode which measures the temperature of the tissues at a definite place and at the exact moment of coagulation. Instead of reading the electric intensity on the milliamperemeter, one notes on the pyrometer the temperature to which the coagulated tissue has been brought. The principles involved

and a description of my pyrometric electrode are given in full detail in a recent article.[1] My method is based on the principle of the electric thermometer. The electrode appears like an ordinary cautery, except that the ball forming the extremity is a plate 2.5 mm. in diameter. It is used just like an ordinary diathermic cautery, but instead of reading the milliamperemeter, one reads only the registration on the pyrometer.

The principal point of my method is that it is not a rapid coagulation, but a slow heating of the sclera, which is the only way to get uniform coagulation. Pyrometers always have a certain degree of inertia, and to know the exact temperature from 20 to 80 C. it is necessary to work slowly for twenty or thirty seconds. With the same time of application and size of electrode, the same effect is always obtained at the same temperature, whatever the conditions may be. Consequently it is possible to study the effects of treatment on the basis of precise anatomic findings in place of clinical approximations. With the pyrometric electrode I have studied in rabbits' eyes the differences in coagulation at different temperatures.

I have explained [2] that scleral diathermocoagulation in rabbits' eyes must be done at 80 C. with a 2.5 mm. electrode, applied for from twenty to thirty seconds. These conditions being realized, the coagulation gives perfect adhesion between the choroid and retina without any damage to the vitreous or to the sclera. The optimum temperature is 80 C.; my experiments have shown that 70 C. is not sufficient, and 90 C. produces too severe reactions. Similar experiments were made on human eyes that were enucleated for sarcoma and the same conclusions were obtained.

I have now operated in fifty cases of detachment of the retina with scleral diathermocoagulation, under exactly the same conditions, i. e., with a 2.5 mm. pyrometric electrode at 80 C. for from twenty to thirty seconds. The results have always been uniform. In the favorable cases, I obtained a cure in practically all instances, with perfect acuity and field of vision, and in the most unfavorable cases improvement occurred.

My operative technic has been fully described in the aforementioned article.[1] The procedure is simple and logical, and certainly gives more security than any other method now in use.

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1. Coppez, Léon: Le traitement du décollement de la rétine par la diathermocoagulation pyrométrique, Arch. internat. de méd. expér. **9**:177 (July) 1934.

2. Coppez, Léon: Le dosage de la diathermocoagulation transclérale par l'électrode pyrométrique, Bull. et mém. Soc. franç. d'opht. **45**:281, 1932; L'utilization de l'électrode pyrométrique dans le traitement du décollement rétinien, ibid. **46**:312, 1933.

SOME OBSERVATIONS AS TO INDICATIONS FOR ADVANCEMENT AND KINDRED OPERATIONS

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Perusal of the prevalent use of advancement, and by that term I include the affiliated procedures of resection, tuck and the shortening operations, will show that they are frequently, if not commonly, used in the following ways:

1. The advancement is often uniformly practiced as the primary and the emphasized operation.

2. Deviation is taken as the main, if not the only, guide to procedure and especially to gradation.

3. One is apt also to be partisan in one's selection of operation and to have a constant leaning to one operation which may be a devisement of one's own or one peculiar to the locality from which it sprang or an institution which has constantly used it and where a student received his learning, and this irrespective of its adaptability.

The theme of this paper is that advancement corrects deviation by producing tension on the muscle operated on and on the deviating muscle, and should be used as the primary operation only when such a type of correction can best be utilized; and that therefore:

1. Advancement while frequently used should not always be made the primary and most emphasized operation.

2. Deviation alone should never be the sole determinator of procedure or gradation.

3. One should exercise a broad selection in choosing the procedure best adapted to the case in question.

Every one recognizes the accomplishments of advancement in the correction of strabismus, but it has a distinctive place and if used improperly it may destabilize end-results. Perhaps the best way to approach an understanding of its use is to go directly to a perusal of the manner by which singly it corrects a deviation.

It has been recognized, but not always, that advancement corrects a deviation by producing tension on the muscle operated on. This is its principal corrective accomplishment. It also adds to its capability of correction by producing tension on the deviating muscle.

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The first corrective agent, i. e., tension on the muscle operated on, pulls the eye toward the nondeviating side. Tension on the deviating side stretches the deviating muscle and diminishes its pull on the deviating side.

This is in direct contradiction to the method by which recession corrects a deviation, which is by bringing about, first, relaxation on the side operated on and second, relaxation on the muscle not operated on.

The very practical observation bearing on the selection of advancement follows that hypertension produced by an advanced muscle will yield a high return of correction if the opposing muscle is not hyperstrong or hypertense, and the equally significant fact that the advancement when opposed by a hyperstrong or hypertense muscle will yield comparatively little correction.

One cannot reiterate often enough that the facts of these opposed muscle strengths point in a large measure as a guide to the selection of procedure. They direct attention to the conditions in which advancement can obtain its largest return of correction, and when it should be the primary or emphasized operative procedure.

In confining oneself to the single operation, the logical conclusion must necessarily be that advancement, when singly performed, will bring about its highest correction when the opposing muscle is not highly muscularized or is not the subject of hypertension.

This is more than emphasized by the fact that the primary effect of a single advancement on the high tension esotropias is comparatively small; on the other hand, when the esotropia is not of the hypertense type, the single advancement gives a high return.

If, then, one had to rely on the single operation for correction one could stop here, simply making the advancement the primary or emphasized operation when a high tension muscle does not oppose it, and the secondary or less emphasized operation when it is opposed by a high-tension, highly muscularized, innervated or spastic muscle.

It may be said that this applies only to the single procedure, but it will be found on careful analysis that the principle of opposing muscle strength, as just enunciated, is the best determinator of which procedure should be the primary or the emphasized feature in the combined operation, and, indeed, as to whether the combined procedure should be undertaken.

Thus, in either event, the first necessity is to ascertain the power underlying the deviation. In the instance of either the single or the combined operation this can be determined by using four tests.

1. The near point of convergence
2. Prism screen test for excess and insufficiency
3. Duction tests

4. What I have so frequently stressed as most important, actual inspection of the exposed anatomy at the time of operation.

A careful computation made and founded on the basis which these tests afford will constitute the rational guide to procedure and will largely support the simple principles of opposing muscle conditions mentioned.

It will also strongly emphasize the error of relying on deviation alone as the guide to procedure and gradation. For it has so often been taken for granted that hypertension or hypermuscularization or innervation necessarily exists on the deviating side, when careful computation would disclose this to be incorrect, and it is a fact that not infrequently there is an unsuspected relative insufficiency on the deviating side.

If this is found to be the case, it will be obvious that advancement should be the primary procedure, for to recede or tenotomize a relatively weak muscle in an esophoria would lead to destabilization, and also it must be evident that the operative effect should not always be divided. When the conditions underlying the deviation disclose a relative insufficiency, advancement, single or bilateral, by reason of its attributes of correction when unopposed by hypertension, should be the primary and probably the only procedure.

On the other hand, in the instance of the high tension esotropias the advancement may well be the secondary operation, if needed at all. In most cases, the opposing muscle strength is far beyond the capability of an advancement to correct, and an unnecessary operation on the attenuated externus is sometimes, especially if the muscle is resected, a mutilation and at most a capsular advancement. And this in some instances prevents the natural recuperation, so often noticeable when tension is removed, of a muscle long subjected to the tension imposed on it by a hypertense opponent. If made the secondary operation in the high tension esotropias, the tendon alone may be resected, leaving all muscle fiber intact.

This illustrates the value of the principle of opposed muscle strengths as a guide to procedure.

There is, however, one group of cases in which at first glance one might say that this principle did not hold good. For in the group of exotropias, advancement of the interni should, as a general rule, be the primary operation. Nevertheless, when the passive quality of the diverging system is recalled and this system is compared with the more highly inergized converging system, the principle of opposing muscle strength and tension is not far amiss, nor is it without value in determining procedure.

Again in the instance of a not particularly hypertense system on one side and a not particularly relaxed system on the other, when one's computation points to the employment of an equal division of the

attribute of advancement in the production of hypertension on one side, and its antithesis, that of relaxation on the other, the division may well take the form of an amount of advancement in millimeters of gradation twice that of recession; for it will be found that while one can advance the externi many more millimeters than one can recede the interni, the recession effect in millimeters on the interni is much more potent in return of correction.

It must be remembered also that the converging system is much more sensitive to operative intervention and should be protected to the greatest extent.

I do not intend to discuss the many different devisements for true advancement and kindred operations. Objections and commendations I feel could be stressed if one were so inclined. But they all have merit, and what is more important is not to confine one's practice wholly to a single procedure when one has at one's disposal a wide range in gradation.

For instance, there are:

1. Operations which leave the muscle end at its normal insertion (resection, tuck and kindred operations) which may be divided into
   - (*a*) Advancement with resection or tuck of tendon alone
   - (*b*) Advancement with resection or tuck of both muscle and tendon
2. True advancements which place the muscle end in front of the insertion, which can be subdivided into
   - (*a*) Advancement without resection of tendon and muscle
   - (*b*) Advancement with resection of tendon alone
   - (*c*) Advancement with resection of tendon and muscle
3. The most extreme procedure, which embodies a muscle advancement and a capsule advancement with a resection of both in conjunction with the outer canthus, all being anchored in mass to the sclerocorneal margin. By this one we can in great measure correct the very marked and unsightly internal paralytic deviations.

Both the selection of the type of operation and the actual gradation in millimeters must be based on the knowledge obtained by painstaking computation of true underlying conditions.

It almost goes without saying that if resection of the tendon alone can be accomplished, leaving all muscle fiber intact, it is of great advantage.

In advancement, as with every other muscle operation, skilful dissection and anatomic readjustment contribute to success. To dissect

a palpably attenuated muscle down to its bare fiber destroys its vascular and nervous supply.

With most of the advancements of this type, the capsule contributes the greatest part of the correction, as a holding medium, rather than the muscle as a contractive agent. Therefore a broad flap of capsule outlined by diverging incisions containing unmutilated muscle, with ligated sutures placed well back and broadly spread, gives all the additional advantage of a properly devised capsular advancement.

I feel the ligated suture just mentioned, which is described in a previously published article,[1] comes as near to an unslippable suture as it is possible to devise, and contributes much to security.

Finally if one recognizes the principle of differentiating opposing muscle strengths, if one does not rely on deviation alone as the sole determinator of procedure or gradation, and if one exercises a broad selection in choosing the procedure best adapted to the case in question, I feel sure one's work on muscles will be attuned to a higher plane.

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1. Jameson, P. Chalmers: The Ligated Suture, Arch. Ophth. **12**:377 (Sept.) 1934.

ASSOCIATION OF OPTIC NEURITIS, FACIAL PARALYSIS AND FACIAL HEMIATROPHY

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In 1904, I [1] presented before the Section on Ophthalmology of the American Medical Association a case observed at the University Hospital, Philadelphia, in the service of Dr. George E. de Schweinitz.

REVIEW OF CASE REPORT

Miss G. W., aged 19, was seen first on Dec. 8, 1903, complaining of blurring of print and aching in the eyes in reading or other close work. Although she was unaware of a loss of sight, examination showed that vision in the right eye was 6/15, and that in the left eye 6/30.

Ophthalmoscopic Examination.—There was atrophy of the optic nerve, more advanced on the left side. The nerve heads were covered with a delicate, white tissue, which blurred their edges and could be traced in the form of white lines along the central vessels of the retina. The veins were of good caliber, but the arteries were reduced to mere threads at the disk, though they were somewhat fuller at a distance from it. The fundus in each eye was otherwise normal. The picture was, therefore, typical of postpapillitic atrophy. The pupils were equal and responded rather sluggishly. The visual fields were moderately contracted, both for form and color, but showed no color scotomas. Under reduced illumination vision in the right eye was only 6/30, and that in the left 2/60. With the photometric test of de Wecker and Masselon the light sense was 4/10 in the right and 1/10 in the left eye. There were, in addition, a flattening of the right side of the face and a decided enophthalmos on this side.

Personal History.—The patient had always been very pale and suffered from irregular menstruation, the amenorrhea lasting sometimes for several months. Of children's diseases, she had had only measles, and had had no recent acute illness of any kind. On Nov. 1, 1902, she consulted Dr. James E. Talley of Philadelphia for paralysis of the right side of the face, which appeared after exposure to a draft and was preceded and accompanied by severe pain in the lower jaw and teeth and in front of the ear on the same side. Dr. Talley found a typical facial paralysis of the peripheral type, but noted no areas of anesthesia. The face was flaccid, the mouth was drawn to the left, and she was unable to close the right eye. There was no ear disease present, but she appeared very anemic and gave the history of menstrual disturbance. Examination of the blood showed: red blood corpuscles, 4,268,000; white blood corpuscles, 5,200, and hemoglobin, 90 per cent. The urine was alkaline, with a specific gravity of 1.022, and contained neither albumin nor sugar; microscopically it was negative, except for phosphates.

Read at the Seventieth Annual Meeting of the American Ophthalmological Society, Lucerne, Canada, July 10, 1934.

1. Shumway, Edward A.: The Association of Optic Neuritis and Facial Paralysis, J. A. M. A. **44**:463 (Feb. 11) 1905.

Treatment.—She was treated with salicylates, strychnine, massage, electricity and later iron. Three weeks afterward, there was some return of motion in the face, and the mouth was straighter, but the eye could not be closed. Early in December 1902 she left the city and was under the care of Dr. R. P. Heilman of Emporium, Pa., who reported that he detected some anesthesia of the right side of the face, but no complaint was made of any disturbance of vision. On her return to the city in March 1903 the motion of the face had become nearly normal, but her mother stated that the right eye was "smaller" at that time than the left, although they had been of the same size before. Dr. Talley saw her in October 1903, and again treated her for irregular menstruation.

Examination.—Dr. William G. Spiller examined the patient. There was stiffness of the right side of the mouth; the eye could be readily closed; there were no areas of anesthesia of the face or conjunctiva; the skin was entirely normal in appearance, but there was a distinct loss of subcutaneous fat which made the face flatter on the right side, and there was a sinking backward of the eyeball. The electrical reactions, tested by Dr. C. S. Potts, were normal, and no weakness of the muscles of mastication could be detected.

Examination in the medical dispensary showed normal lungs, slight extension upward of the heart, dulness on the left of the sternum, and a soft systolic murmur on lying down, heard best at about the third interspace, just to the left of the sternum, which disappeared on standing, and was probably hemic. The pulse was rapid but regular. Examination of the blood showed: red blood corpuscles, 4,248,000; white blood corpuscles, 13,500, and hemoglobin, 58 per cent. The urine was slightly cloudy, with a specific gravity of 1.030, showed no albumin or sugar, and contained numerous epithelial cells but no casts.

In discussing this case I said:

In 1894 Hoffmann [2] described two cases of facial paralysis, associated with low-grade optic neuritis, retrobulbar in type, with central scotomas for colors, and considered them as cases of multiple neuritis due to cold. He said that optic neuritis was very rarely associated with facial paralysis, but that Gowers had seen three cases of rheumatic optic neuritis in women, two of whom had suffered from neuritis of the facial nerve and other rheumatic affections previously. De Schweinitz [3] reported two cases in 1899, in which the optic neuritis, which was retrobulbar in type, with central scotomas, occurred two years after an attack of facial paralysis. In the first case, both eyes were affected, but not at the same time; in the second, there had been several attacks of sclerokeratitis, two attacks of right facial paralysis, and finally left-sided optic neuritis. Leszynsky [4] reported a case before the New York Neurological Society in 1900, in which during an attack of facial paralysis, severe pain appeared in the supraorbital and temporal regions on the left side, which lasted about one week, when the patient became blind in this eye; the ophthalmoscopic examination revealed a well-marked papillitis. He attributed the optic neuritis to periostitis in the optic canal, so that the condition was a multiple neuritis, probably due to rheumatism, although syphilis could not be positively excluded.

These 7 cases added to the present one, make a total of only 8 reported; the association is, therefore, quite rare. A number of cases of optic neuritis, however,

2. Hoffmann, J.: Deutsche Ztschr. f. Nervenh. **1**:72, 1894.
3. De Schweinitz, G. E.: J. Nerv. & Ment. Dis., May 1899, p. 263.
4. Leszynsky, W. M.: J. Nerv. & Ment. Dis., March 1900, p. 150.

has been reported in polyneuritis. According to Remak [5] in Nothnagel's System, it has been found by E. Remak, Goldscheider and Fuchs in spontaneous (infectious) polyneuritis; by Schanz in puerperal forms; with carcinoma (autointoxication) by Miura; in alcoholic polyneuritis by Lillienfeld, Giese and Pagenstecher, and Jolly. Other cases have been reported since Remak's article was written. For example, Wetz, in his inaugural dissertation, Giessen, in 1903, gives a case of bilateral recurring retrobulbar neuritis, appearing at long intervals associated with polyneuritis. But even the number of such cases is limited, and, with but few exceptions, the neuritis is retrobulbar in type, in both series.

A second case of facial paralysis with partial hemiatrophy of the face was also reported, which occurred in a patient of Dr. William G. Spiller of Philadelphia, with the following history [6]:

The patient, Mrs. Rosa K., aged 52, suffered from peripheral right sided facial paralysis after exposure to cold in July 1902. At the time of the attack she had severe pain on this side of the face. On examination in February 1904, nineteen months afterward, the paralysis had disappeared except for slight stiffness of the face, but there was a decided flattening of this side, and distinct enophthalmus. The eyes, however, showed no ophthalmoscopic changes, and vision was normal in each eye, with the proper correction of a low refractive error.

The question of the causation of the enophthalmos and flattening of the face was also discussed, and Gowers' opinion in his textbook was quoted that "muscular wasting follows the nerve degeneration in facial paralysis, in the face, as elsewhere, and in those who have but little subcutaneous fat, the loss of tissue may be perceptible; but the nutrition of the subcutaneous tissues is unaffected and *true facial hemiatrophy* never results from disease of the facial nerve." No cases of true hemiatrophy following facial paralysis could be found at that time in the literature. In summing up I said:

1. Optic neuritis is occasionally associated with facial paralysis, either alone, or as part of a multiple neuritis; the etiologic factor may be rheumatism, *but at times appears to be an infection, the nature of which is as yet undetermined.* The optic neuritis is usually of the retrobulbar type, but a decided papillitis may be present, and be followed by more or less marked atrophy. In cases of multiple neuritis, of the cranial nerves, the eye-grounds should be examined for possible optic nerve complication.

2. In facial paralysis, flattening of the face and enophthalmus may appear, and are to be considered as due to a neuritis of the fifth nerve, and not to involvement of possible sensory fibers in the facial nerve.

CONTINUATION OF CASE REPORT

The subsequent history of the first case, however, seemed to me to warrant calling attention to the subject again, particularly to the etiology,

5. Remak and Flatow, in Nothnagel: Specielle Pathologie und Therapie Vienna, vol. 2, p. 111; full bibliography given here.

6. Shumway,[1] p. 465.

the more because of later developments in the views of neurologists and physiologists as to the functions of the facial nerve.

In 1909, seven years after the first attack of facial paralysis, the patient had oculomotor paralysis; in 1912 she had a recurrence of facial paralysis, and another recurrence in 1919 on the *left* side; six months later the *right* side was involved. On the last occasion she was under the care of Dr. D. Clinton Guthrie of Philadelphia, who thought that the right third and sixth nerves were also involved. By this time the importance of infection from local foci about the mouth was becoming well understood by the medical and dental professions, and a roentgen study of the patient's teeth was made by Dr. George Parry, a dentist of Philadelphia, who reported that the left superior central incisor tooth, which had been filled with gold when she was 13 years old, with death of the nerve, showed a large irritated abscess, and on its removal a long pus sac, or dentigerous cyst, "as large as the end of the thumb" was found, which extended upward to the level of the floor of the orbit and the ethmoid sinus. An abscess was also found connected with a left lower molar, which was also extracted.

In 1920, fifteen years after the paper was written, examination in my office showed weakness of the right side of the face, with twitchings of the eyelids on both sides. The right palpebral fissure was slightly narrower than the left. *Touch and temperature sensations* were equal on the two sides of the face. There was no sense of smell, and little sense of taste on the anterior part of the tongue. Both optic nerves showed white atrophy, with blurring of the disk margins from old neuritis; the arteries were narrowed and the dilated veins were clustered like a bunch of earthworms on the nerve heads. The retinas showed no lesions, and the patient was without perception of light.

Despite her blindness, the young woman completed her education in the Pennsylvania Institution for the Instruction of the Blind, and became an accomplished pianist. Some years later she fell out of a window, apparently accidentally, and was killed. There was no autopsy.

COMMENT

In view of the complete history, I think that there can be no question that this was a case of recurrent polyneuritis, which involved the optic nerve, causing atrophy and blindness, and the third, fifth and seventh (possibly also the fourth and sixth) cranial nerves. Whether the loss of smell and taste was due to involvement, respectively, of the *first* and *ninth* (*glossopharyngeal*) *nerves* may be questioned and will be discussed later. The etiology seems to point with a fair degree of certainty to a focus of infection in the abscessed teeth.

It is of great interest that in Remak's monumental volume, "Neuritis and Polyneuritis," in Nothnagel's "Specielle Pathologie und Therapie," which was published in Vienna in 1900, the most important factor in the etiology of peripheral neuritis was considered to be chilling (*Erkältung;* in 45 per cent, according to Remak; in 70 per cent, according to Bernhardt). Other less important factors were (1) trauma, (2) auto-intoxications, (3) dyscrasias, such as gout, rheumatism, carcinomatosis and diabetes, (4) intoxications such as chronic poisoning by

alcohol, lead, arsenic and carbon monoxide, (5) arteriosclerosis and (6) infections such as typhoid, typhus, smallpox, scarlatina, measles, leprosy, syphilis (constitutional), diphtheria, whooping cough, follicular angina, erpsipelas, pneumonia, mumps, cerebrospinal meningitis, malaria, influenza and beriberi.

Only a short chapter was devoted to the consideration of infections (bacterial) in the etiology of mononeuritis and polyneuritis, which have since been considered of so great importance that it appears to me the pendulum has perhaps swung too far in this direction, and one is too prone in careless taking of the history to think only of a focal infection and neglect other possible systemic diseases.

Finally as to the functions of the seventh nerve: This is not the place to discuss in great detail the complexity of the nerve fibers composing the seventh nerve, which is really a combined motor and sensory nerve, with the geniculate ganglion, as the origin of its sensory fibers, situated on the nerve, during its passage through the facial or fallopian canal. One of its motor nerve bundles arises from a nucleus in the floor of the fourth ventricle and controls the closure of the eyelids; it is so close to the nucleus of the third nerve that it may be considered as part of this nucleus. The other main bundle arises in the pons, close to the nucleus of the sixth nerve. The many anastomoses with the fifth and glossopharyngeal nerves increase its complexity. The anatomy and physiology may be found in great detail in Landois'[7] "Textbook of Human Physiology." The studies of Hunt[8] in connection with herpes of the ear, facial paralysis and disturbances of the auditory nerve in 1907 and 1910, which symptoms are now accepted generally as the "Hunt syndrome," have further clarified the views of neurologists as to the functions of the seventh nerve (Aitken and Brain[9]). In the present instance it may be accepted, I think, that the facial paralysis was due to a facial neuritis, and that the atrophy of the face was not a true hemiatrophy (which usually occurs in early life) and was due to inflammation of the fifth nerve. The third (and possibly also the fourth and sixth) were likewise involved. As to the disturbance of smell, this may possibly have been due, as Landois said, to involvement of the facial nerve, either by interference with the dilatation of the nasal orifice from weak-

7. Landois: Text-Book of Human Physiology, ed. 10, edited by A. F. Brubaker, translated by A. A. Eshner, Philadelphia, P. Blakiston's Son & Co., 1904, p. 694.

8. Hunt, J. R.: Herpetic Inflammations, J. Nerv. & Ment. Dis. **34**:73, 1907; The Symptom-Complex of the Acute Posterior Poliomyelitis of the Geniculate, Auditory, Glossopharyngeal and Pneumogastric Ganglia, Arch. Int. Med. **5**:631 (June) 1910.

9. Aitken, R. S., and Brain, R. T.: Facial Palsy and Infection with Zoster Virus, Lancet **1**:19 (Jan.) 1933.

ness of the levator nasi or, more likely, by defective distribution of tears (in consequence of paralysis of winking and of the muscle of Horner), which leaves the corresponding side of the nasal cavity dryer than normal. As both facial nerves were affected, at different times, entire loss of smell may in this way be accounted for. The loss of taste may also be assumed to be due to disease of the facial nerve, by involvement of the fibers constituting the chorda tympani branch, which come from the glossopharyngeal nucleus, without the necessity of considering involvement of the glossopharyngeal nerve itself, especially in view of the absence of disturbance of the soft palate.

SUMMARY

I have reported the case of a young woman with postneuritic optic atrophy, recurrent attacks of facial palsy, some of which were preceded by pain, partial hemiatrophy and enophthalmos of one side of the face, loss of smell and partial loss of taste.

The diagnosis was: polyneuritis of cranial nerves probably due to dental infection. Consideration has been given to the complexity of the seventh nerve and to the disturbances of sensation from its inflammation because of involvement of its own sensory tract, which arises in the geniculate ganglion, and also of the anastomosing fibers from the trigeminal and glossopharyngeal nuclei. Brief mention has been made of Hunt's syndrome, viz., occurrence of facial paralysis, vertigo, tinnitus, deafness (at times temporary, occasionally permanent) with herpes of the external auditory canal and anterior part of the auricle, from inflammation of the geniculate ganglion in the facial canal and involvement of the facial and auditory nerves in this position.

HYDROGEN ION CONCENTRATION OF TEARS

ITS RELATION TO CERTAIN OCULAR SYMPTOMS AND TO CONJUNCTIVAL AND CORNEAL LESIONS

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At present the treatment of conjunctival and corneal lesions is almost entirely symptomatic and empirical and, in many instances, thoroughly illogical. Two years ago one of us (G. N. H.) called attention to the frequent misuse of the ointment of yellow mercuric oxide (Pagenstecher's ointment)[1] and stated that much of its reputation for improving all sorts of conjunctival, corneal and tarsal lesions for which it was not originally intended was probably gained in the days when it was prepared by the apothecaries from mercuric chloride and potassium hydroxide. It is highly probable that the washing of the precipitated oxide was often incomplete and that traces of free alkali remained in the amorphous powder after it was dried. Probably with equal frequency there was an excess of mercuric chloride left, and when this went into solution in the tears a decidedly acid reaction resulted. (The p_H of a 1:10,000 solution, freshly prepared, is 5.2. The p_H of a 1:20,000 solution is 5.3.)

Attention was called to the well known fact that the presence of free acid or alkali profoundly affects the bacterial flora, and it was suggested that these early salves might have had an inhibiting effect on the bacterial growth in any given case because of their content of acid or alkali. It is, of course, equally probable that the alteration in the reaction was favorable to the growth of the organisms and hence detrimental to healing. The oxide itself is probably too slightly soluble to have any significant chemical effect, although the physical properties of the oxide produce unpleasant sensations and are inimical to healing except where the abrasive action is desired for a brief period, as in phlyctenular keratitis.

Presented before the Eye, Ear, Nose and Throat Section of the California Medical Association, at the Sixty-Third Annual Session, Riverside, Calif., May 3, 1934.

1. Hosford, G. N., and McKenney, J. P.: Ointment of Yellow Mercuric Oxide (Pagenstecher's Ointment), J.A.M.A. **100**:17 (Jan. 7) 1933.

Striking evidence of the importance of the hydrogen ion concentration in ocular symptoms is so common that it has escaped our notice and the notice of generations of our predecessors. We doubt if there is a single member of this society who has not listened many times daily to Mrs. "A's" account of how she cured her "cold in the eye" with boric acid and Mrs. "B's" account of how the same panacea made her "cold in the eye" worse.

It is true that these observations are usually mingled with the patient's description of his own and his ancestors' visual prowess, and this reiterated circumstantiality becomes profoundly fatiguing. Nevertheless, we shall always regret that these narratives did not lead us to study the subject of the p_H of the tears years ago.

It was not until the appearance of the work of Gifford and Smith [2] in 1933 that the problem engaged our active interest. These investigators have rendered a great service to English-speaking ophthalmologists by emphasizing the work of Lipschütz [3] of Berlin, who showed that the sensations experienced by a patient when solutions are dropped into the conjunctival sac or on the cornea are much more intimately related to the hydrogen ion concentration of the solution than to its osmotic pressure.

In the past when ophthalmologists have given this matter any thought, they have assumed that the disagreeable sensations were due to disturbances of the osmotic pressure. To mitigate the unpleasant symptoms, we naturally reduced the concentration of the drug and increased the frequency of the doses, often without relieving the disagreeable immediate effects.

Many have found Ringer's solution a more agreeable and satisfactory medium than distilled water, but few have realized that the kind of glass out of which the bottle is made and the liquid in which it is washed have more to do with the immediate sensation produced in the patient's eye than the composition of the vehicle or the therapeutic agent which the vehicle contained. Naturally, when a patient requires but a single dose, the sensation it produces is relatively unimportant, but whether or not the sensation is disagreeable becomes preeminently important when the dose must be repeated frequently over long periods of time as with physostigmine, for example. The effects on the conjunctival and corneal tissue of solutions which depart too far from a p_H which corresponds to that of the tears will be considered later, but a tribute is due these structures for the way in which they withstand all manner of chemical insults for protracted periods when they are in

2. Gifford, S. R., and Smith, R. D.: Effect of Reaction on Ophthalmic Solutions, Arch. Ophth. **9**:227 (Feb.) 1933.

3. Lipschütz, H.: Ueber die Bedeutung der Wasserstoffionen Konzentration im Bindehautsack, Klin. Monatsbl. f. Augenh. **82**:763 (June 21) 1929.

a state of comparative health. Most patients in the more sophisticated centers of population feel obliged to wash their eyes with something (usually a saturated solution of boric acid, undisguised or under a pseudonym) at least once a day, and it is much easier and more agreeable for the oculist to lend tacit approval to the practice than to discourage it. Nevertheless, we believe the practice to be illogical and at times detrimental and shall attempt to point out the reasons later.

HISTORY OF HYDROGEN ION DETERMINATIONS OF THE TEARS

Dr. C. F. Charlton [4] of Pasadena is given credit by both Duke-Elder [5] and Adler [6] in their recent textbooks for the first determination of the hydrogen ion concentration of the tears. His results were published in 1921, and the figure he obtained by the colorimetric method was p_H 7.2. The following year a Japanese, Kusonocki, is credited by Duke-Elder with obtaining the value from 7.4 to 8.4. His method was not stated in the available abstract of his work.

In 1927 Professors Oguchi and Nakashima [7] of the University of Nagoya published in German an extensive investigation of the p_H of tears in health and in disease. Professor Oguchi was particularly well qualified to undertake an investigation of the subject, as he had previously addressed himself to the investigation of the cytology and bacteriology of the conjunctiva. He undertook to determine the p_H of tears and ocular secretions separately and also investigated the p_H of solutions of the various drugs used in the treatment of diseases of the eyes. He selected the colorimetric method as the most practical and used the standard buffer solutions of Sörensen and Clark and the indicators thymol blue, phenol red and methyl red. He used what he called a "spotting method" in which a platinum loop full of material was mixed with a loop (presumably the same loop) full of indicator on a clean white glass plate, and compared the color obtained with that produced by a loop full of indicator mixed with a loop full of buffer solution of known p_H. He recognized the presence of certain errors introduced by salts and albumin in such a method, but correctly considered them too small to be serious. A more important error, viz., the mass action of the indicator itself in such a concentration, was disregarded. We believe, therefore, that while his results have relative

4. Charlton, C. F.: Protein in Tears and Innervation and Secretion of the Lacrimal Gland, Am. J. Ophth. **4**:647 (Sept.) 1921.

5. Duke-Elder, W. S.: Textbook of Ophthalmology, London, Henry Kimpton, 1932, p. 640.

6. Adler, Francis: Clinical Physiology of the Eye, New York, The Macmillan Company, 1933, p. 19.

7. Oguchi, C., and Nakashima, M.: Ueber die H-Ionenkonzentration der Augensekrete und der Augentropfen, Arch. f. Ophth. **119**:300, 1927.

validity, his figures must not be taken as absolute. Oguchi also placed his greatest emphasis on the secretions obtained after the tears had been carefully removed from the conjunctival sac with Japanese lens paper. This also seems to be a mistake from the point of view of future therapy directed toward altering the p_H, as under clinical conditions the p_H of the tears themselves mixed with the secretions must be considered, because it is in this form that they are always in intimate contact with conjunctival and corneal lesions. In the seventeen cases in which the tears were not wiped away, the result in a variety of diseases was a p_H of 7.4, a value which coincides closely with what we have come to regard as normal for the tears of healthy eyes in people who are free from symptoms. His results in this group of cases did not exhibit the variation and deviation from that value which we have found in pathologic conditions. He concluded that by his methods the tears themselves are quite alkaline, between p_H 8.4 and p_H 8.6, and properly stated that this is too alkaline for most bacterial growth, but we have not found the p_H of the tears as high as this except in the case of vernal catarrh.

Lipschütz [3] stated that the p_H of normal tears is 8, but did not tell how that value was obtained. He stated further that he made an approximation of the p_H of the tears in various types of conjunctivitis and found it to be about 7.5. His method consisted of dropping dilute neutral red on the everted lid and comparing the color obtained with the color produced by dropping the same quantity of neutral red into an undesignated amount of standard phosphate buffer solution of known p_H. He was aware of the obvious errors of such procedure. (The chief errors are, first, the difference in the color of the background, which is the eyelid in one case and some container for the standard in the other; second, the difference in the thickness of the films of colored fluid which are compared, and third, the incomplete dissemination of the indicator.) He pointed out one important source of error in all determinations of the p_H of tears, namely, the rapid loss of the dissolved carbon dioxide into the air. This rapidly shifts the reading several tenths toward the alkaline side.

In 1930 von Pelláthy and Schneider [8] published another method of estimating the p_H of the tears. They dipped strips of filter paper impregnated with indicators directly into the tears between the tarsal and the bulbar conjunctiva; then, on a plate of white glass, they compared the color obtained with that of a series of similarly prepared papers dipped in buffer solutions of known p_H. This method has the

8. von Pelláthy, B., and Schneider, K.: Behandlung von Bindehaut-und Hornhautentzündungen, besonders von Trachon mit Natrium hydrocarbonicum auf Grund von Untersuchungen über p_H-Wert des Konjunktivalsekretes, Klin. Monatsbl. f. Augenh. **85**:774 (Dec.) 1930.

advantage that it tests the p_H of the actual fluid in contact with the conjunctiva and the cornea, but it is subject to many errors. Paper is far from uniform in its own reaction, the amount of indicator varies, and, in addition to the rapid loss of carbon dioxide, the papers rapidly begin to dry, thus modifying the color obtained. New standards would have to be made for each test. The authors expressed the belief that the test should be made without stimulating a flow of tears and stated that a rapid flow of tears shifts the result to the alkaline side. They found the p_H of tears in sound eyes to be from 7.6 to 8. In conjunctivitis and corneal ulcer, it was about 8.

Thus we see that the results obtained by different observers vary considerably and are subject to a number of errors. Any determination will have to be made by a method that requires only a small quantity of tears, and it is probable that stimulation produces a more alkaline tear, or a tear of altered chemical composition.

Dr. J. Howard Brown's [8a] colorimetric method of determining the p_H of small quantities of liquid seemed to have certain advantages over the foregoing methods provided the composition of the tears is such that they are sufficiently buffered to permit some dilution without altering their p_H. In general, dilution with distilled water tends to minimize the errors introduced by the alcohol in which the indicators are dissolved, the mass action of the indicators themselves and the protein and salt errors which will be discussed presently. The change from body temperature to room temperature modifies the reading several hundredths of a p_H, but as all determinations in this paper on tears and buffer solutions were carried out at (or very near) 25 C., the p_H at body temperature can be calculated if desired.

DEGREE OF BUFFERING OF TEARS

With the Brown apparatus it is easy to demonstrate that the tears ars sufficiently buffered to permit a dilution of 1:15 with neutral distilled water without altering the p_H.

The demonstration of the degree of buffering of the tears was made in the following way:

Five drops of freshly boiled and cooled neutral distilled water were placed in each of seven of the small glass cells of the apparatus. Tears were then collected in a pipet, care being taken not to aerate them. To one empty cell and to the first cell which contained water, 5 drops of tears were added. The cell which contained the tears and the water was gently agitated, and 5 drops of the mixture were then transferred to the third cell. The third cell was agitated, and 5 drops of this mixture were transferred to the fourth cell. This procedure of serial dilution was repeated until eight cells had been prepared. This gave a series com-

8a. Brown, J. H.: The Colorimetric Determination of the Hydrogen Ion Concentration of Small Amounts of Fluid, J. Lab. & Clin. Med. **9**:239 (Jan.) 1924.

prising, first, undiluted tears, then dilutions of 1:1, 1:3, 1:7, 1:15, 1:31, 1:63 and 1:127 and ending with a dilution of 1:255. Exactly 1 drop of alcoholic indicator was added to the undiluted tears and to each of the dilutions as rapidly as they were made. The color obtained was constant until after the fifth cell (dilution 1:15) was reached. In the sixth cell (dilution 1:31) there was a marked change in color which indicated the end of the buffer effect and the beginning of what would be expected in an ordinary unbuffered solution.

Sufficient tears for this experiment can be obtained only under abnormal conditions, but there is no reason to believe that the results will be materially different if enough tears can be obtained without accidental or artificial stimulation of the lacrimal gland. We may, therefore, safely conclude that a dilution of 1:5 is within the range where buffer action is not exhausted, and as 6 drops of fluid are adequate for a satisfactory reading with this apparatus, we may assume that the reading which we obtain represents a close approximation of the true p_H. It is almost always possible to obtain 1 drop of tears without undue stimulation, and the mass action of the indicator will play a smaller part in a mixture of 1:5 than when lower dilutions are used as in the "spotting method."

PROTEIN ERROR

The effects of the various constituents of the tears on the p_H must be considered. According to the published analyses of tears, the proteins are made up of albumin, 0.394 per cent, and globulin, 0.275 per cent. The shift in the p_H produced by these substances is in opposite directions, and when the tears are diluted five times the concentration of albumin and globulin is such that the shift is less than plus or minus 0.03[9] and is therefore negligible when phenol red is used as an indicator.

SALT ERROR

The concentration of salts in the tears totals somewhere between 1 and 1.3 per cent and is made up principally of sodium and potassium chlorides and phosphates. These salts introduce a small error into the reading of the hydrogen ion concentration as determined by an indicator.

When phenol red is used as the indicator, this salt error amounts to less than plus or minus 0.15.[10] It is apparently slightly greater with bromthymol blue, and data are not available for cresol red, but most tears will, under clinical conditions, have a p_H that lies within the range where phenol red is useful.

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9. Martin, C. J., and Lepper, E. H.: Protein Error in Estimating p_H with Neutral Red and Phenol Red, Biochem. J. **21**:356, 1927.

10. Clark, W. M.: The Determination of Hydrogen Ions, ed. 3, Baltimore, Williams & Wilkins Company, p. 181.

Since the concentration of tears used in testing is approximately the same in all cases, the salt error will likewise be essentially the same, and therefore our results will contain a small but relatively constant error which, however, will always be in the same direction.

The method used has certain advantages over the other methods which have been used heretofore. It may be quickly applied and is reasonably accurate. The greatest sources of error are the rapid loss of carbon dioxide from the tears and the effect of temperature. The light used for matching the colors must be uniform and should approximate daylight of a uniform intensity and direction.

By this method the p_H of tears collected with as little stimulation as possible from the lower culdesac closely approximates that of the blood, viz., p_H 7.35, although the same persons will exhibit some variation at different times and yet be free from all ocular symptoms.

The difficulty of correlating the p_H of the tears with such symptoms as itching, burning, smarting and photophobia is greater than we at first supposed. Patients vary so much in their accounts of their sensations that perhaps a correlation should not even be attempted, although we believe these symptoms are, if not solely dependent on the p_H, at least intimately associated with it. It may safely be stated that it is unusual for patients to be altogether comfortable if the p_H, as determined by the foregoing method, is above 7.8 or below 6.6, and these are about the limits of the range of the p_H of solutions that can be dropped into the conjunctival sac without exciting unpleasant sensations if the p_H of the tears is approximately normal before the solutions are instilled.

THERAPEUTIC EFFECT OF BUFFERED SOLUTIONS

As one would expect, if the reaction of the tears is already highly alkaline and an acid buffer solution is instilled, relief is experienced. The relief afforded is different from that produced by cocaine in that it is practically instantaneous and often complete, but naturally of shorter duration.

Injuries to the cornea are uniformly painful if the ophthalmic branch of the fifth nerve and its central connections are intact, and, almost without exception, produce a marked shift of the p_H of the tears to the alkaline side (from p_H 8 to p_H 8.4). While lacrimation is stimulated in the uninjured eye, the p_H does not rise a corresponding amount and often remains at 7.3 or 7.4. Ulcers of the cornea, if acute and painful, also produce a shift to the alkaline side, and an acid buffer solution affords considerable relief, although it is not so marked as in the case of injury.

Vernal catarrh produces a marked shift in the p_H of the tears toward the alkaline side (from p_H 8 to p_H 8.4). It is so marked that

all investigators have noted it no matter what method of determining the p_H they have used, and we can confirm the observation, although only one case has come to our attention since this investigation was started. This alteration accounts for the empirical observation, which was made long before the p_H of bodily secretions was investigated, that dilute acetic acid frequently affords relief from symptoms although it does not cure the disease. For therapeutic use in such cases an acid buffer solution has several theoretical advantages which simple solutions of even weakly dissociated acids do not possess. It will neutralize the excess of alkali as efficiently, but will not produce the unpleasant symptoms characteristic of free acids of a p_H below 6.6, and it probably does not stimulate the production of more alkali.

On the other hand, chronic ulcers, particularly in trachoma, usually do not produce this alkaline shift. This was the basis on which von Pelláthy [11] and later Luzsa [12] advocated the use of sodium bicarbonate in the treatment of trachoma and its complications.

There is no agreement in the literature on the p_H of the tears in either acute or chronic conjunctivitis, and in our own series we have found deviations both to the alkaline and to the acid side of what we consider normal. Patients with conjunctivitis come to the oculist after a great variety of home remedies have been tried, and the p_H obtained in these cases is the resultant of the p_H of the tears and of the medicaments instilled before the patient left home.

The duration of the effect of the instillation of several drops of buffer solution is as yet unknown to us except so far as it may be inferred from the symptoms. It must depend on several factors, one of which is the buffer capacity of the solution used. This can readily be determined by titration and must be kept at the maximum in the preparation and handling of the solutions. Another is the rate at which tears are being secreted. This apparently tends to diminish and approximate normal under the influence of the appropriate buffer solution, although no accurate means of measuring the flow suggests itself. A third factor is the actual content of acid or alkali of the tears, the titration of which presents great difficulty. A fourth is the configuration and muscular tone of the lids. Clinically, the symptoms serve well enough as a guide to the frequency of the dose. The duration of the relief from symptoms varies from a few minutes to several hours, and the degree of relief also varies with the character of the lesion and the temperament of the patient.

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11. von Pelláthy, A.: Heilversuche mit Natriumhydrocarbonicum in der Augenheilkunde, Klin. Monatsbl. f. Augenh. **79**:163 (Sept. 2) 1927.

12. Luzsa: Ueber die Behandlung der Augenkrankheiten mit Natriumhydrokarbonat. Klin. Monatsbl. f. Augenh. **87**:769. 1931.

It has been our policy to use a buffer solution or a therapeutic agent that will tend to bring the p_H toward 7.3 when there is a marked deviation from normal and the causative organism is unknown or a mixed infection is present. The tolerance of certain organisms to variations in the p_H is limited. The pneumococcus, for example, cannot live in a medium the p_H of which is below 7.[13] It seems logical, therefore, to use an acid medicament, and as 1 per cent optochin hydrochloride solution has a p_H of 6.6 this may well be a factor in its usefulness against the pneumococcus. The results have been gratifying at times, but again healing has apparently not been accelerated nor have the symptoms been ameliorated.

Staphylococci and streptococci will tolerate wide variations in the p_H of the medium in which they are growing but tend to form acid, so it is perhaps logical to use an alkaline buffer solution as there is no specific substance that can be counted on to kill them in a concentration that can be safely used in the eye.

The gonococcus probably has a much greater tolerance to alterations in the p_H in vivo than it has in vitro, although the reported limits are wide even here (from p_H 5.8 to p_H 8.2). The urine, for example, will show greater variations than this, so that the prospects of killing or inhibiting the growth of this organism in the body by altering the hydrogen ion concentration are not great. It is well to remember in treating gonorrheal ophthalmia that, in general, weak acid solutions tend to harden the cell membranes and weak alkaline solutions tend to soften them. Silver nitrate solution has a decidedly acid reaction.

In chronic conjunctivitis, the alteration of the p_H of the tears by the instillations of buffered solutions may be of great value. It is probable, but as far as we know, not proved, that the presence of acid may stimulate the production of a more alkaline tear and that the presence of alkali may stimulate a more acid tear, as is the case with the saliva. The histologic similarity of the lacrimal gland and the parotid gland suggests such a possibility, although the experimental proof would be exceedingly difficult on account of the anatomy of the region. The multitude of ducts from which the lacrimal fluid is poured over the eye makes it practically impossible to collect the tears at their source.

It seems almost essential to postulate such a beneficent, reciprocal, chemical mechanism, or it would not be possible to pour saturated boric acid into the conjunctival sac day after day without doing some damage. In fact, such practices in the aged and debilitated are capable of setting up inflammation, and when such patients can be persuaded to give up the use of boric acid the lids frequently exhibit prompt improvement. At any rate, it seems thoroughly illogical to pour acid on an inflamed

13. Buchanan, R. E., and Fulmer, E. I.: Physiology and Biochemistry of Bacteria, Baltimore, Williams & Wilkins Company, 1930, vol. 2, p. 315.

surface, or at least to do it repeatedly. Frequently in these cases the p_H of the tears is low, perhaps from the exhaustion of the aforementioned reciprocal mechanism or from conditions of the body as a whole of a more general character. Even if the tears are alkaline it is therapeutically preferable to use a buffered solution with a p_H slightly below the neutral point rather than a solution containing 4 per cent of acid and with a p_H of 5.2. The principal reason that it can be done without obvious damage is because boric acid is but slightly dissociated and is washed out by the tears before all the hydrogen ions instilled can become active.

It is unfortunately not practical to hand over the formulas worked out by Gifford and Smith to the average druggist and get solutions of the desired p_H. The factors of error in making them up under such conditions are too great. Further, their formulas were published, not as the final or best buffered solutions, but merely as an improvement on what we had been using as vehicles and not as therapeutic agents in themselves. The alteration of the hydrogen ion concentration of the tears, however, cannot fail to be of importance both in healing and in the production and control of symptoms apart from all other considerations.

Our own plan has been to work with a single buffer system at a time. The one which suggested itself first was a boric acid-sodium tetraborate system. This offers a range in p_H from 6.6 to 9.2 [14] and is fairly satisfactory. This system, although obviously safe and harmless, introduces a foreign polyvalent ion into the tears, the antagonistic effects of which on the monovalent sodium and potassium ions is impossible to predict with certainty and which may have some effect of its own apart from the buffer action of the hydrogen and hydroxide radicals which accompany it, and with which we are more concerned in the present investigation.

The carbonates would be ideal except for their instability, but this renders them practically useless for this purpose because the p_H of a carbonate system will change almost overnight.

At present we are using fifteenth-molar phosphate buffer solutions with a p_H of 6.6 on the acid side and a p_H of 7.6 on the alkaline side. This is probably an advantage, as the total salt content is not much above the point where the lytic action of lysozyme is greatest. Solutions with a greater buffer capacity can readily be made and should probably be given a trial, but when enough sodium chloride is added to the fifteenth-molar phosphate buffer solutions to bring them to isotonicity with the tears the relief of symptoms is definitely less and the thera-

14. McClendon, J. F., and Medes, Grace: Physical Chemistry in Biology and Medicine, Philadelphia, W. B. Saunders Company, 1925.

peutic results are not so satisfactory. It may be possible to devise a single buffer solution of p_H 7.3 with enough capacity to neutralize any deviation the tears may exhibit either to the acid or the alkaline side of normal. If this can be done satisfactorily, it would do away with the necessity for determining the p_H of the tears in each individual case (except for purposes of investigation). A careful study of the Hofmeister series may suggest many different buffer systems, some of which are certain to be more effective in particular situations than others.

It may be possible to devise substances of low solubility which can be dusted into the conjunctival sac and which may exert a more prolonged effect than it is possible to obtain from solutions which must be instilled, but the physical stimuli would probably tend to vitiate their possible chemical effects in most instances. Certain amphoteric colloids may have possibilities.

Our experience with the keeping qualities of the various buffer solutions which we have tried has been entirely different from that reported by Gifford and Smith, and before buffer solutions can be put to widespread therapeutic use some type of preservative which does not interfere with their action will have to be added. To do so now would introduce another variable into an already complex problem which is by no means solved.

The alkaloids employed in ophthalmology are usually used in the form of solutions of salts of one of the strong acids, for example, sulphates, hydrochlorides or nitrates. When these salts go into solution they produce a p_H well to the acid side of neutral, and even when they are dissolved in a buffered solution of p_H 7.6 the buffer capacity may readily be exhausted and a fairly acid solution remain. It would, therefore, seem advisable to make a study of the available salts of the alkaloids commonly used and select those the solutions of which are nearest to p_H 7.3 or those in which the solution can be readily adjusted to this point or near it. Solutions of the salts of the alkaloids are not always stable at p_H 7.3; in fact, some are not even soluble there. This point may not in all cases correspond to the point at which the drug is most effective, so that one must choose judiciously between effectiveness and lack of irritating qualities.

In using buffered solutions for therapeutic effects, we do not know with certainty what p_H we should aim to produce, but the optimum p_H for the growth of epithelium and connective tissue may indicate what p_H we should attempt to approximate in conjunctival and corneal lesions. Fischer [15] has shown that the optimum p_H for the growth of fibroblasts is from 7.4 to 7.8, and that a slight change above or below these limits

15. Fischer, Albert: Growth of Fibroblasts and Hydrogen Ion Concentration of Medium, J. Exper. Med. **34**:447 (Nov.) 1921.

materially retards growth. Fibroblasts grow for only from four to six generations at a p_H of 5.5 and not much over ten generations at a p_H of 8.5.

It is probable that epithelium has a greater tolerance to alterations in the p_H, but satisfactory experimental work on this point was not found in the literature.

The question of how much one should alter the hydrogen ion concentration of the tears (given the power to do so) is, of course, of paramount importance. It is apparent that one must relieve symptoms when possible, but not at the expense of inhibiting regeneration of tissue or interfering with the action of the lytic enzyme, lysozyme, which is probably the most important factor in the natural defense of the ocular apparatus against bacteria.

SUMMARY

1. A synopsis of previous work on the determination of the hydrogen ion concentration of the tears is presented.

2. The clinical application to the tears of the Brown apparatus (LaMotte) for the determination of the hydrogen ion concentration of small quantities of fluid is described.

3. It has been shown that the tears are sufficiently buffered to permit the degree of dilution necessary to render the determination of the hydrogen ion concentration easily applicable to clinical cases.

4. The average hydrogen ion concentration of tears from healthy eyes which were free from symptoms closely approximates the p_H of the blood, viz., a p_H of 7.35.

5. The protein and salt errors are discussed.

6. Lipschütz' observation that "the sensations experienced by a patient when solutions are dropped into the conjunctival sac or on the cornea are much more intimately related to the p_H of the solution than to its osmotic pressure" is confirmed, and success in the relief of some conjunctival and corneal symptoms by the modification of the p_H of the tears by the instillation of appropriate buffer solutions is described.

7. The relation of the p_H of the tears to certain bacteria and to the healing of conjunctival and corneal lesions is discussed.

Professors Chauncey D. Leake of the department of pharmacology, University of California Medical School, T. C. Daniels and C. F. Allen of the California College of Pharmacy, University of California, Dr. D. M. Ervin and E. R. Andrews of San Francisco and Dr. F. L. LaMotte of Baltimore gave valuable aid in this investigation.

HISTOLOGIC OBSERVATIONS ON THE FUNDUS IN LEUKEMIA

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The microscopic structure of the choroid and retina in the various forms of leukemia has been carefully described by many competent observers, whose reports have in turn been reviewed by Leber,[1] Kümmel[2] and Hudson.[3] The present study, consisting of a histologic examination of the globes in eleven cases of leukemia, indicates the frequency of typical and atypical leukemic lesions in general and draws attention to certain features rarely met with in previous studies.

Of the group under study, three were cases of chronic myeloid leukemia, five of acute myeloid leukemia and two of chronic lymphatic leukemia. In all ten cases, the clinical diagnosis was confirmed by autopsy. The clinical diagnosis in still another case was aleukemic lymphadenosis, and autopsy revealed diffuse hyperplasia of the lymph nodes and lymphatic infiltrations in several internal organs. Ophthalmoscopic examination had been done in nine of the eleven cases and revealed positive findings in all but one (chronic lymphatic leukemia). A constant feature was the presence of retinal hemorrhages and edema of the disk or blurring of its margins. Retinal exudates occurred in two of the cases, but so-called leukemic retinitis, consisting of a leukocytic infiltration of the retina, especially along the veins, and associated with hemorrhages, was found only once (case 10).

Evidences of previous bleeding in the retina were noted microscopically in all the cases. Accumulations of leukocytes unrelated to retinal blood vessels were seen frequently. The white patches bordered with hemorrhages, noted during life, presented surprisingly little indication of their presence histologically; red blood cells, hypertrophy of the nerve fiber layer and disturbance in the nuclear layers (cystic

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1. Leber: Die Erkrankungen der Netzhaut bei Leukämie und Pseudoleukämie, in Graefe-Saemisch: Handbuch der gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, 1916, vol. 7, p. 973.

2. Kümmel, R.: Arch. f. Ophth. **95**:105, 1918.

3. Hudson, A. C.: Roy. London Ophth. Hosp. Rep. **18**:153, 1911.

spaces) were the only marks of an exudative process. Dilatation of the retinal vessels, increase in the number of leukocytes in their lumen and slight edema of the disk were almost constant features. Extravasation of leukocytes into the sheaths of the vessels was seen in only one case (case 11). In the choroid, slight overfilling of the blood vessels with leukocytes was the rule. In three cases, the entire choroid was slightly thickened because of the presence of large numbers of white cells in its blood vessels and a slight degree of extravasation. Case 4 showed isolated deposits of white blood cells about one of the distended choroidal vessels. Marked increase in the thickness of the choroid because of leukocytic infiltration occurred only once, in case 10. Two cases were so unusual in their clinical and pathologic manifestations as to deserve special attention.

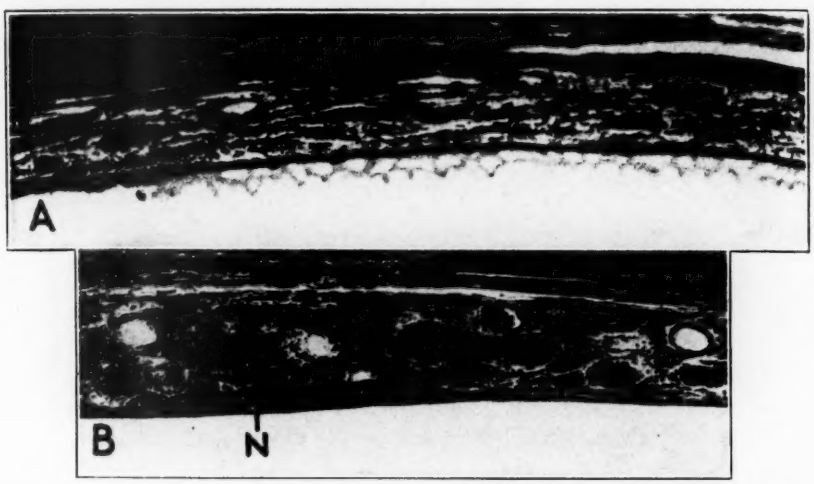

Fig. 1.—In *A*, the choroid is slightly thickened. The blood vessels are distended with leukocytes. In *B*, in addition to the dilated blood vessels, there is a nodular collection of leukocytes; *N*, in the choroid.

REPORT OF CASES

CASE 10.—The clinical diagnosis in the case of S. P., a white woman of 28, was acute myeloid leukemia. The blood count was as follows: hemoglobin, 43 per cent; red blood cells, 2,300,000; white blood cells, 44,000, of which 89 per cent were myeloblasts. In the fundi, the margins of the disk were indistinct and the veins dilated, but there were no white spots or hemorrhages. Chemosis of the bulbar conjunctiva suddenly developed in the left eye, with slight exophthalmos. Edema of the orbit was suspected, but orbital hemorrhage could not be ruled out, although there was no ecchymosis of the lids. The blood count on the following day showed the hemoglobin to be 23 per cent, while the white cell count had risen to 240,000. Numerous hemorrhages now appeared in the fundi and increased in size and number until death ten days later.

The most striking feature in the microscopic examination of the left eye was the thickening of the choroid to four or five times its normal size. This dimension was uniform up to the region of the equator of the globe, from which point it tapered off gradually toward the ora serrata. The layer was packed with cells of the large myeloblastic variety to such an extent that the intervening walls of

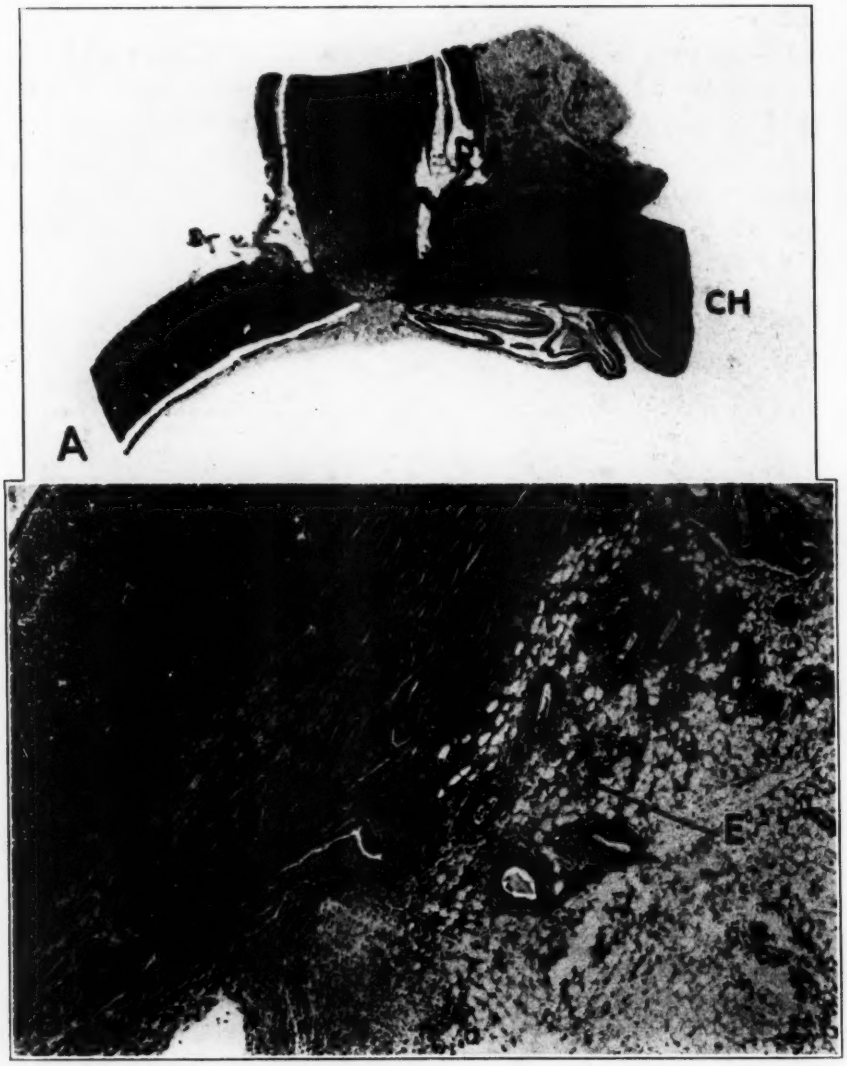

Fig. 2.—In *A*, the choroid is densely infiltrated and hypertrophied to a uniform width up to the region of the equator. In *B*, the ciliary vessels, *C*, and the episcleral vessels, *E*, are enmeshed in leukocytes. *CH* is the choroid.

the blood vessels were reduced to thin streaks. These appeared to be broken up to allow the escape of cells into the intervascular space. Pigment cells were sparse. The choriocapillaris layer was obliterated. The posterior ciliary vessels in their course through the sclera were encased in a mesh of leukocytes resem-

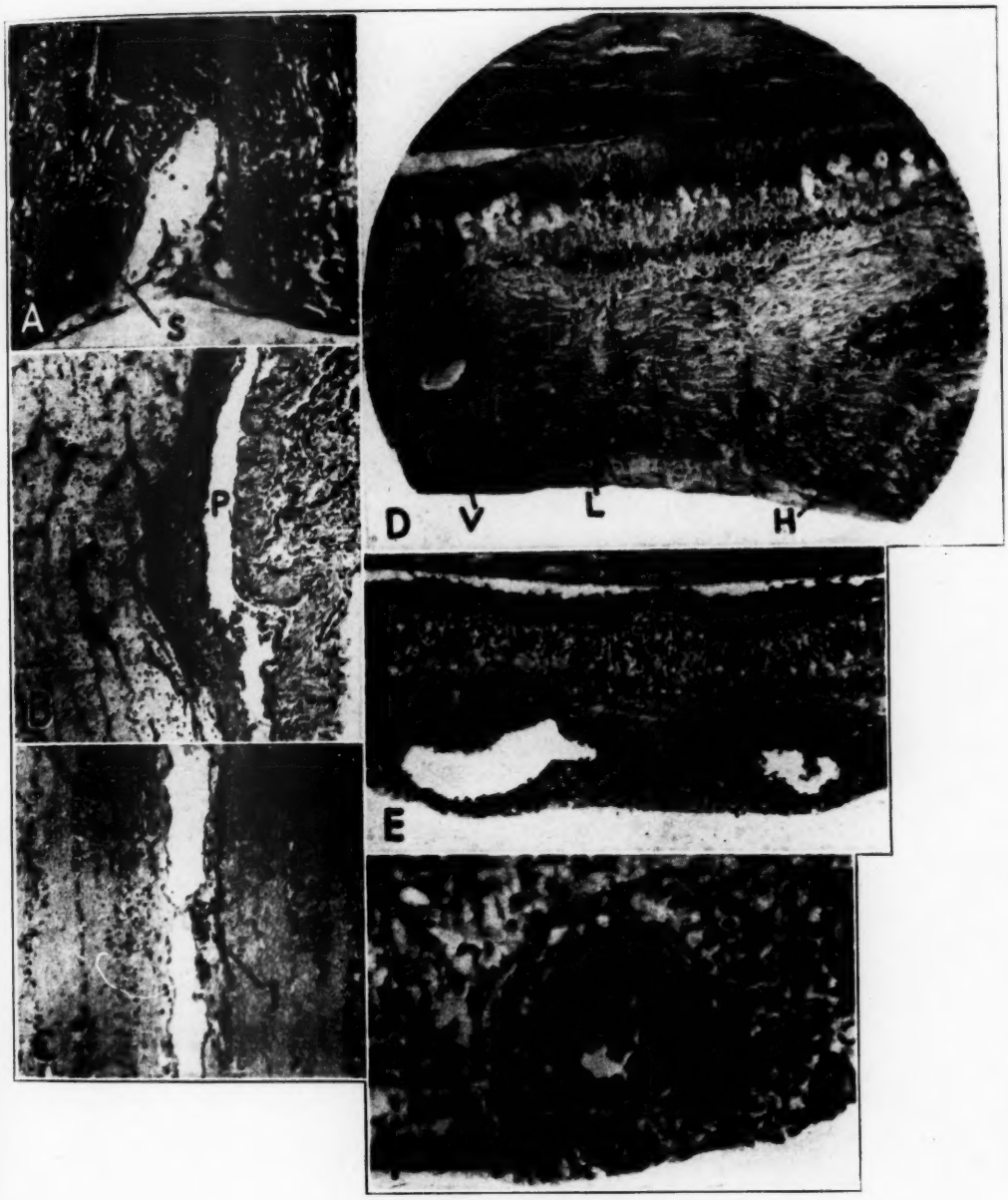

Fig. 3.—In *A*, the disk is edematous and infiltrated. *S* is a dense collar of cells about a retinal vein. In *B*, the pia and the septums of the optic nerve are infiltrated with leukocytes. In *C*, at *1*, the intima of the central retinal vein contains numerous white blood cells. *D* shows a section close to the disk. *V* indicates a thickened vein; *L*, leukocytes in the nerve fiber layer, and *H*, hypertrophied nerve fibers. In *E*, both retinal blood vessels are dilated and contain parietal thrombi. The retina is densely infiltrated with red blood cells and leukocytes. *F*, a retinal arteriole in cross-section, shows necrosis of the wall of the arteriole and separation of its fibers by blood cells.

bling those seen in the choroid. The episclera was similarly infiltrated, the cells being especially prominent about the ciliary vessels. The disk and retina were edematous and otherwise showed little to distinguish the case from the others studied.

CASE 11.—S. F., aged 9 years, had been ill only two weeks when a diagnosis of acute myeloblastic leukemia was made. The blood count was as follows: hemoglobin, 45 per cent; red blood cells, 2,590,000; white blood cells, 1,070,000, of which 94 per cent were of the monocytic type, probably myeloblasts. The fundi showed both disks swollen from 3 to 4 diopters, their margins obscured by patches of exudates. These appeared to be extravasations of white cells rather than edema. They proceeded for some distance into the retina and were prominent along the veins, which were greatly swollen. The entire retina was soaked with blood to an extent seen only in thrombosis of the central retinal vein. The right eye was secured for examination following death, which occurred after an illness of six weeks.

Microscopic examination showed that the disk contained a large number of white blood cells. At its edges the veins were sheathed by densely grouped leukocytes. The lumen of the vessel in each instance was markedly narrowed. At many points in the nerve there were aggregations of leukocytes in the pia and in the septal fibers close to the pia. Of peculiar interest was the presence of small groups of similar cells immediately beneath the endothelial layer of the central retinal vein in its course through the nerve. These occurred in several foci along the course of the vein.

In sections near the disk the leukocytic infiltration around the veins persisted and smaller sheaths about the arterioles appeared. The adjoining retina contained many similar scattered cells. The nuclear layers were quite disturbed. The walls of the large venules were distended with edema and showed more than the normal number of nuclei. The nerve fibers, especially those close to the disk, were distinctly hypertrophied. Where evidences of bleeding in the retina were most marked, the veins were markedly dilated and their lumen occupied by portions of fibrinous blood clot which was adherent to the wall of the vessel. The wall itself was so densely infiltrated by white and red blood cells that its normal elements were hardly discernible.

At such points where the wall of the vessel was not so studded with cells, it was seen that fragmentation and separation of its fibers had occurred. The fibers themselves had undergone necrosis.

COMMENT

From this study of eleven eyes in cases of leukemia it appears that in most instances the retinal lesions are minimal, since they do not produce profound disturbance in the retinal structure, and the retinal vessels are normal in appearance. Such changes as marked sheathing of the veins, together with exudation of large numbers of leukocytes into the retina and nerve, are decidedly the exception. Also, fragmentation and necrosis of the walls of the larger vessels must be unusual. Similarly, in the choroid, while dilatation of the sinuses with leukocytes to a variable degree is the rule, the enormous infiltration with enlargement noted in one of the cases is exceptional.

The observation of the destruction of the wall of the blood vessel following infiltration of its wall by leukocytes is not new. It was noted by Murakami [4] who offered it as the explanation of the escape of the blood elements into the retina. In his case, solution of the walls of the blood vessels had proceeded to the point where it had entirely disappeared. Other vessels showed "aneurismal and ampulliform enlargements" as a result of "thrombotic varicosities." In the present case, in

Clinical Data in Cases in Which Eyes Were Studied Microscopically

Case	Patient	Age	Clinical Diagnosis	Total Leukocyte Count	Hemoglobin, per Cent	Ophthalmoscopic Examination of the Fundus
1	S. J.	60	Chronic lymphatic leukemia	14,000	12-25	Disk margins blurred by edema; hemorrhages throughout retina
2	S. K.	32	Acute myeloid leukemia	24,800	55	Disks blurred; white spots on disks; scattered hemorrhages
3	O. S.	22	Chronic myeloid leukemia	250,000	60	Recent and old exudates on disk; retinal and preretinal hemorrhages
4	B. W.	61	Aleukemic lymphadenosis (autopsy, leukemic lymph adenoma)	22,000	50	Fundus not examined
5	M. K.	36	Chronic myeloid leukemia	268,800	60	Hemorrhages on disk and in retina
6	S. K.	4½	Acute myeloid leukemia	12,000	55	Edema of disk; preretinal and few retinal hemorrhages
7	O. P.	7	Acute myeloid leukemia	93,000	51	Disk edges blurred; few linear retinal hemorrhages
8	S. R.	33	Chronic myeloid leukemia	52,000	70	Fundi not examined owing to scarring of corneas
9	A. K.	48	Chronic lymphatic leukemia	725,000	68	Fundus examined with negative results
10	S. P.	28	Acute myeloid leukemia	44,000 (before death 240,000)	43 (before death 23)	Some peripapillary edema; few hemorrhages before death
11	S. F.	11	Acute myeloid leukemia	1,070,000	45	From 3 to 4 diopters papilledema; large patches of exudates along veins; retina soaked with blood

which the fragments of the wall of the vessel were necrotic, the only explanation is invasion or local proliferation of blood cells.

Of unusual interest in this study was the sheathing of the posterior ciliary vessels and the infiltration of large numbers of leukocytes into the episclera, both in association with the infiltration into the choroid. In this case exophthalmos and edema of the conjunctiva of the left eye had occurred more or less coincidentally with a marked increase in the white blood cell count. Borgeson and Wagener [5] stated that diffuse infiltration of the choroid is usually seen in association with lymphoma

4. Murakami, J.: Klin. Monatsbl. f. Augenh. **39**:136, 1901.
5. Borgeson, E. J., and Wagener, H. P.: Am. J. M. Sc. **177**:663, 1929.

of the orbit. They further pointed out that such an infiltration is apparently due to local extravascular proliferation of immature leukocytes, since it has been found in cases in which the total number of leukocytes in the blood was not increased. These authors quoted Hudson, who considered the orbital lymphoma as the probable primary lesion, while Meller,[6] on the other hand, believed that it arises secondarily by extension from the choroid along the sheaths of the posterior ciliary vessels. While the orbital tissues were not examined in the present case and orbital lymphoma therefore not demonstrated, it is fair to assume, from the condition of the episclera, that such a lesion existed and was responsible for the edema and the exophthalmos. While it is possible that the sudden increase in the number of white blood cells was a factor in its etiology, the microscopic appearances do not warrant a definite statement as to whether the extra-ocular infiltration came by way of the choroid or directly from the blood stream.

6. Meller: Arch. f. Ophth. **62**:130, 1906.

LECTURES ON MOTOR ANOMALIES OF THE EYES

II. PARALYSIS OF INDIVIDUAL EYE MUSCLES

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If in a case of orthophoria a single muscle becomes paretic, deviation in the primary position is always present, being latent or manifest according to the intensity of the paresis and the function of the paretic muscle. A latent or only temporarily manifest deviation results if, for instance, the internal rectus is weakened, since paretic divergence of not too high a degree can be overcome by a convergence impulse. But even the slightest paresis of one of the elevators or depressors cannot be kept latent, because the vertical range of fusion is very small. That is true only of cases of orthophoria. In a case of heterophoria the paretic deviation is modified by the anomalous position of rest—increased or diminished, or even completely abolished or compounded of various components, as will be shown presently in discussing the palsies of individual muscles.

In any case of paresis the position of rest must be altered by the loss of tonus of the paretic muscle and the intact tonus of the other muscles. If this is the only reason for the deviations taking place, the deviation will disappear and the former orthophoria will be restored as soon as the paresis is cured, i. e., as soon as the paretic muscle has regained its normal tonus. This cannot happen if the paretic deviation is caused not only by the normal tonus of the intact muscles but by the development of a secondary contracture of the antagonist of the paretic muscle, due to a change of its structure. In such cases a part of the deviation will remain even when the paresis is cured. In any case of typical paresis of a single muscle the angle of deviation increases or decreases according to whether the movement of the eye is in the direction of action of the paretic muscle or of its antagonist. The exceptions to this rule I shall discuss shortly.

The diagnosis of a muscular palsy can easily be made if the movement of the paretic eye in a certain direction is limited. But in many cases of slight paresis there is no such limitation, because the limits of the ocular movements depend not only on the intact function of the eye muscles but also considerably on the fascial check ligaments. In order to reach the normal limits of movement in the various directions a maximum innervation is not necessary. A moderate innervation is enough to bring the eyes to the normal limits, which the check ligaments do not allow to be exceeded. Therefore, even a paretic muscle can

bring the eye to the normal limit by means of an excessive innervation. Hence the uniocular field of fixation is not of nearly so great value for the diagnosis of a slight paresis as is the binocular field of fixation or the field of binocular single vision, the limits of which are nearly identical.

According to Hering's law of the invariably equal innervation of both eyes, a motor impulse must produce a different effect on each eye if one of the muscles executing the bilateral movements is paretic. From this a considerable limitation of the binocular field of fixation must result, even if both eyes tested separately are able to reach the normal limits of movement. The most important signs of the typical palsies, for instance, the difference between the primary and the secondary deviation, and its increase and decrease in looking in the direction of action of the paralyzed muscle or its antagonist, derive from the aforementioned fundamental law of association of the movements of the eyes. The consideration of these signs and symptoms may be omitted at this point, as I shall come back to them in discussing the atypical palsies. Only a few rules of general validity need be considered before I turn to the special forms of paralyses.

ABDUCENS NERVE PARALYSIS

In many cases of palsy an *anomalous position of the head* is a characteristic sign. This is explained by many authors as an endeavor to make up for the function of the paralyzed or weakened muscle; it is obtained by turning the head in the direction in which the eye muscle had been acting before it became paralyzed. This explanation is not invariably correct even for the external and internal rectus muscles, much less for the elevators and depressors. True, in most cases of palsy of the abducens nerve there is a habitual turning of the head to the side corresponding to the direction of action of the paretic muscle; but figure 1 shows photographs of a patient with abducens nerve palsy holding the chin pressed against the chest. This position of the head helped the patient to binocular single vision, whereas if the head was thrown back, so that the eyes had to look down, the pathologic convergence present in the primary position of the head was increased to a high degree. Why did the patient not turn the head to the side of the paralyzed abducens nerve? Because of the long duration of the palsy, secondary contracture of the internal rectus had set in, and the deviation could not be overcome by looking toward the sound side, as it could be in the early stages of the palsy, when it was small and uncomplicated. On the other hand, it is a physiologic rule that the elevation of the eyes favors divergence, just as the eyes are disposed to converge in looking down. Both tendencies are due to the mechanical factors determining the position of the eyes. With few exceptions, one finds

in cases of orthophoria latent divergence when the eyes are looking up and latent convergence when they are looking down. Therefore, in cases of slight or of moderate convergence in the primary position of the eyes, the deviation may disappear if the patient looks up or lowers the head, whereas it is increased either by looking down or by throwing the head back. The reverse is true in cases of latent or of manifest divergence.

Much more frequently than in cases of paresis of the external or internal rectus the rule under discussion concerning the so-called vicarious rotations of the head turns out to be inaccurate in palsies of a single elevator or depressor muscle. It is valid only if both elevators or both depressors of one or both eyes are paralyzed; in such cases the head is rotated around the frontal axis, i. e., thrown back or depressed. But in cases of trochlear nerve palsy the position of the head is seldom in

Fig. 1.—*A* shows the habitual anomalous position of the head in a case of inveterate abducens nerve palsy. The patient while holding the chin pressed against the chest has binocular vision. *B* shows the occurrence of pathologic convergence while the head is thrown back. Most of the illustrations in this paper (figs. 1 to 17) are reproduced from Bielschowsky, A.: Die Lähmungen der Augenmuskeln, in Graefe and Saemisch: Handbuch der gesamten Augenheilkunde, edition 2.

accordance with this rule. Usually the head is tilted toward the shoulder of the sound side and at the same time turned around on the vertical axis so that the paretic eye is rotated outward. In cases of very slight paresis the position of the head will show only one of the components just mentioned. Another example is that in cases of slight paresis of the right superior rectus muscle the face is often turned only to the right, so that the paretic eye is adducted. How can such a position of the head effect binocular single vision if an elevator muscle is paretic? It is known that the vertical rectus muscles have most influence on the vertical movements when the visual line is abducted so that it lies within the muscular plane. The more it moves away from this plane,

the more do those muscles lose their influence as elevators and depressors, whereas simultaneously the influence of the oblique muscles on the vertical movements increases in nearly the same proportion. Therefore, the vertical deviation due to the paresis of the right superior rectus decreases and at last disappears if in consequence of turning the head to the right the paretic eye is adducted.

The rule concerning the vicarious rotation of the head must be modified as follows: The patient chooses the least inconvenient position of the head by which the paretic muscle is sufficiently disburdened so that binocular single vision can be obtained.

If a general practitioner is consulted by a patient because of diplopia of paretic origin, he will not infrequently bandage the sound eye, taking it for the paralyzed one. This mistake is due to the patient's constantly using the paretic eye for fixation. There are several reasons for a secondary deviation taking place at the beginning of the paralysis: first, that the vision of the paretic eye is better, the nonparetic eye being amblyopic; second, that the patient is much disturbed by diplopia, the distance of the two images being small in primary deviation whereas the distance is much greater in secondary deviation and therefore the diplopia is much less disturbing, and finally, that some patients prefer fixation with the paretic eye because it has been dominant since childhood.

Another symptom of ocular palsy, called false orientation, is interesting from a scientific point of view although one can dispense with it for diagnosis. If the patient while covering his sound eye is asked to point toward an object situated in front of him or in the field governed by the paralyzed muscle, the finger will point more or less to the side of the object and toward the field of action of the paretic muscle. The more the muscle is paralyzed, the more the pointing finger will deviate from the correct direction. The origin of this mistake is obvious: The absolute or egocentric localization of objects in space is nearly correct as long as the correlation between the innervation of the eye muscles and its effect is correct, i. e., as long as the amount of the actual movement corresponds to that which is intended. But if the right eye is deviated inward owing to a paresis of the external rectus muscle and one asks the patient while covering his left eye to fix an object in the median plane of his head, he has to turn the paretic eye to the right, and from this he will get the idea that the object to be fixed is lying in the right half of the visual field, so that his finger points to the right instead of to the median plane. On measuring the amount of the error by a method more exact than the primitive touch test, one will find that it coincides with the secondary deviation; i. e., the visual axis of the covered, sound eye is directed to the point at which the paretic eye localizes the fixed object. This behavior is found

only in cases of a recent palsy. If the patient is forced to use the paretic eye continually for fixation, the absolute localization will soon adapt itself to the changed relations between innervation and its effect, and the false orientation becomes rectified. But then one will find a different kind of false localization in testing the nonparetic eye while the paretic eye is covered. The patient will point wrongly, but to the opposite side, that is, to the left, if the left eye, which usually is in secondary deviation, is induced to fix. This kind of false localization is called spastic, in contradistinction to the paretic localization just discussed. Spastic localization is the term for the opposite disproportion between motor innervation and its effect after the innervation in such a case has become adapted to the paresis of the fixing eye, it is stronger than is required by the normal muscles for the given amount of movement.

Fig. 2.—*A* shows a very small primary deviation in a case of complete left abducens nerve paralysis. *B* shows an enormous secondary deviation.

The extent of the paretic deviation varies individually within wide limits without being proportionate to the degree of the palsy. The photographs (fig. 2) of a person with complete abducens nerve paralysis show a very small primary deviation (*A*) and an enormous secondary deviation (*B*). In contrast to this, numerous patients with slight paresis display a considerable, even a very high, degree of primary deviation from the beginning of the illness. These remarkable differences are due partly to the individual differences in general muscle tonus. In cases of unusually small paretic deviation I have found the tendon reflexes unusually weak; on the other hand, in cases of strikingly high deviation the reflexes have been abnormally increased. Neurologic examination has confirmed my assumption of an unusually weak or an unusually increased muscle tonus, respectively. But I do not believe that the degree of paretic deviation depends wholly on the general muscle tonus.

Possibly the region and the kind of lesion causing the paresis also influence the degree of the deviation. If a lesion in the nuclear region produces paralysis of the external rectus muscle and at the same time affects the fibers transmitting the impulse of inhibition to the internal rectus, so that it cannot relax, one may assume that the paretic deviation will be higher than it would be in a case of basal abducens nerve palsy, in which the inhibitory impulses going to the internal rectus are undisturbed. But this theory still requires proof by further research.

Among the most characteristic signs of the palsies belong the difference between the primary and the secondary deviation and also the increase and decrease of the angle of deviation according to whether the gaze is directed to the field of action of the paralyzed muscle or to the opposite side. It is not unusual that in a case, for instance, of abducens nerve palsy which is quite typical in the beginning the difference between the primary and the secondary deviation decreases more or less gradually and at last disappears, the deviation thus becoming no longer distinguishable from a concomitant squint or from a divergence paralysis, particularly as even in looking toward the side of the paralyzed eye the angle of deviation increases just as little as it decreases in looking toward the other side. This behavior can be explained in the following manner: If the paresis of the external rectus muscle is getting better, while at the same time a contracture of the internal rectus is developing, the deviation does not decrease but extends gradually over the whole field of fixation. In spite of the fact that the external rectus has regained its normal function, the deviation remains by virtue of the contracture of the internal rectus. This contracture, if it is only a functional one sustained by nervous influence, can disappear in course of time, and binocular single vision can be restored in the whole field of fixation. Or the contracture can become permanent by a gradual change of the muscular structure of the internal rectus. Then a concomitant deviation remains, the angle of squint being approximately equal in the whole field of fixation. I am sure that some cases of the latter kind have been taken incorrectly for divergence paralysis, which I shall discuss later.

In the great majority of cases of abducens nerve palsy there is the usual homonymous diplopia. Sometimes, however, one encounters a case which displays all the symptoms of an abducens nerve palsy with the sole exception that the homonymous double images are not on the same level and that they are a slightly inclined toward one another. This may be due to a complication of the abducens nerve palsy by a concomitant hyperphoria or by paresis of one of the vertical motors, or the position of the external rectus may not be symmetrical with the horizontal meridian of the eye. One may assume the last mentioned factor to be the cause if the vertical and the rotatory components of

the deviation are rather small in comparison with the horizontal component, and if only the latter increases or decreases in the typical manner according to the direction of the gaze. The other components of the deviation represent a subordinate (secondary) function of the paretic muscle and its antagonists, due to their asymmetrical adjustment to the eyeball.

TROCHLEAR NERVE PARALYSIS

By far the most frequent and most important type of paralysis of a single vertical motor is palsy of the trochlear nerve—most important because there are not infrequently variations leading to a wrong diagnosis. The first striking sign in many cases is habitual torticollis, i. e., a tilting of the head toward one shoulder. The ocular origin of the torticollis is often not recognized, especially in cases of congenital trochlear nerve palsy or those acquired in early childhood. The general practitioner or the surgeon who is first consulted about the torticollis frequently takes it for a contracture of the sternocleidomastoid muscle, although there is neither a contracture which can be felt nor a resistance against the passive straightening of the head or against its being tilted toward the opposite side.

I have observed many such cases in children who had to endure various kinds of orthopedic treatment for several years, naturally without the least effect. As soon as the physicians or the parents discontinued the forced straightening of the child's head, it was tilted toward the same side as before the treatment. At last the physician advised the parents either to punish the child because of the bad habit or to divide the sternocleidomastoid muscle. But when the operation was done, the child did not cease to tilt the head in the same way as before. In several cases the child's mother was the first to discover the ocular origin of the position of the head by observing that the child closed one eye during the forced upright posture of the head, whereas both eyes were opened as soon as the child was allowed to keep the head tilted in the habitual way. This correct observation is easy to understand: The habitual position of the head helps the child to secure binocular single vision, whereas to avoid a disturbing diplopia arising from straightening the head the child closes one eye. These children lose the anomalous position of the head spontaneously as soon as the balance of the vertical motors of the eyes is restored by the required operation, which I shall discuss later.

The ocular origin of this kind of torticollis was first recognized by Cuignet,[1] in 1873; he could not, however, explain the connection any more than could Landolt,[2] in 1890, in his paper on ocular torticollis.

1. Cuignet: Torticollis oculaire, Rec. d'opht., 1873.
2. Landolt: Torticollis oculaire, Bull. méd., Paris **4**:573, 1890.

Without knowing the problem under discussion, A. Nagel,[3] in 1871, had supposed that in cases of slight paresis of an elevator or a depressor muscle a vertical and rotary deviation would be caused by tilting the patient's head toward one side, a supposition based on the discovery that a parallel rotation of the eyes around the visual axis is produced by tilting the head toward the opposite side. That parallel rotary movement had to be performed, as Nagel presumed, by both the inferior muscles of the one eye and at the same time by both the superior muscles of the other eye. The combined action of the two superior muscles as well as that of the two inferior muscles cannot cause a deviation of one of the visual lines provided the two muscles of each pair are equally strong, for in that case the antagonistic components will compensate each other and there can result neither a vertical nor a lateral deviation of the visual axis. The only effect of the combined action of those two muscles is the rotary movement which they produce in the same direction. Let one suppose that in a case of right trochlear nerve palsy the head is tilted toward the right shoulder. From this will arise a vestibular excitation of the vertical motor muscles, which are able to produce a parallel rotary movement of the eyes to the left. This movement is produced in the left eye by the two inferior muscles, and in the right eye, by the two superior muscles. The paralyzed right superior oblique muscle cannot compensate any more the elevating and the adducting component of the superior rectus, from which a vertical and lateral deviation of the right visual line must result, whereas in a normal person both the visual axes would be stationary. And what will happen if the head of the patient with the paretic right superior oblique muscle is tilted toward the left side? Both inferior muscles of the right eye and the superior muscles of the left eye receive the vestibular innervation to rotate the eyes around the visual axis to the right. This movement can be performed without the cooperation of the paretic muscle; no deviation of the visual axis will result. As I mentioned before, the more the sound muscles are burdened the more favored will be the paretic muscle. Now one can understand the reason for the habitual tilting of the head that is observed in so many cases of trochlear nerve palsy: If the head is tilted toward the shoulder of the sound side, the paretic superior oblique muscle is favored, so that binocular single vision is obtained.

Nagel's supposition has been proved correct by the investigations made by Hofmann and me [4] in 1900. In that publication the explanation given by other authors concerning the tilting of the head in cases of

3. Nagel, A.: Ueber das Vorkommen von wahren Rollungen der Augen um die Gesichtslinie, Arch. f. Ophth. **17**:243, 1871.

4. Hofmann, F. B., and Bielschowsky, A.: Die Verwertung der Kopfneigung zur Diagnostik von Augenmuskellähmungen, Arch. f. Ophth. **51**:174, 1900.

trochlear nerve palsy was discussed and proved wrong. But, strange to say, that wrong explanation has found currency to this day in many later articles on the subject. It is said that the paretic eye, deviated upward by tilting the head, is depressed as far as necessary to bring both of the visual axes to the same level, so that the vertical distance between the double images is removed and only a small lateral distance remains, which can be easily corrected by a convergence or divergence innervation. The error of this explanation can be recognized at once. If one puts a prism of 10 degrees, base up or down, before one eye and tilts the head alternately toward either side, one will see the two images at the same height, but only in respect to the horizontal plane, so that if one makes an effort to converge the double images pass each other without meeting. The direction in which they are moving will deviate from the direction in which one sees them moving with the head erect by the same angle which the basal line of the eyes, i. e., the line between both the nodal points, includes with the horizontal.

There is another argument against the wrong explanation just mentioned: In cases of concomitant vertical divergence or of paresis of the superior or inferior rectus muscle the tilting of the head does not influence the distance between the two images. Therefore, this peculiar posture of the head will be found only if one of the oblique muscles is paralyzed and the patient can get binocular single vision by tilting the head.

I have not yet been able to find out why the tilting is not of any use in palsies of the vertical recti, although these muscles help to bring about rotation of the eyes around the visual axes necessitated by tilting the head toward the opposite side. But according to my experience, one may assume that paresis of the superior or inferior oblique is in all probability the cause of the habitual tilting of the head, provided that only by this posture is binocular single vision to be obtained.

It may be stated, in brief, that in some cases of trochlear nerve palsy the habitual position of the head differs from the one just described because the patient chooses the most convenient position of the head which relieves the paretic muscle enough to permit binocular single vision. Cases will be encountered in which the head is turned toward the sound side, so that the visual line of the paretic eye, because of being abducted, is not acted on by the oblique muscles. A habitually depressed position of the head in cases of trochlear nerve palsy is seldom met with.

For an exact investigation of the influence which the position of the head just discussed exerts in certain cases of vertical deviation, one may use a simple apparatus constructed on the principle of Helmholtz's *Visierzeichen* (fig. 3). While the patient's head is fixed by his taking between his teeth the little plate at one end of the rod, he looks at a

horizontal black strip on a piece of white cardboard fixed to the other end of the rod 30 inches (75 cm.) away. The rod is put through a short tube, so that when the patient tilts his head it rotates around the same axis and through the same angle as the head. In this way it is insured that the visual line keeps its direction during the tilting of the head, since the cardboard with the fixed strip keeps pace with the movement of the head, in respect to both the amount and the direction. A patient with a left trochlear nerve palsy using this little apparatus will see, while his head is erect, two images of the black strip, the image belonging to the left eye being below the other image and both converg-

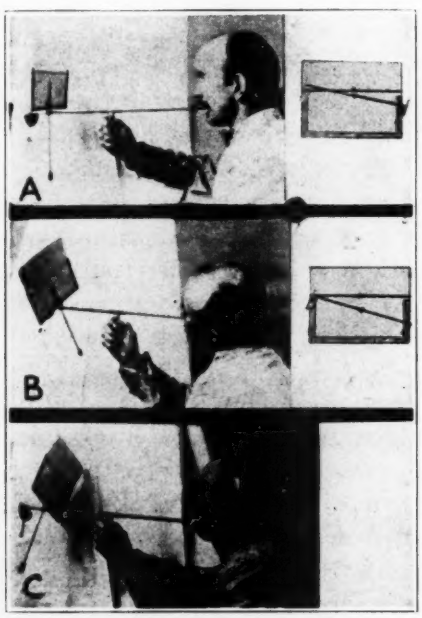

Fig. 3.—Head-tilting test in a case of left trochlear nerve palsy. In *A*, while the patient's head is erect two images of the fixed black strip are seen (reproduced on the right side of the photograph). In *B*, the vertical distance between the two images is increased by tilting the head toward the left shoulder. In *C*, tilting the head toward the right shoulder helps to secure binocular single vision.

ing toward the left side (*A*). When his head is tilted toward the left shoulder, the vertical distance and the obliquity will increase considerably (*B*), whereas tilting of the head toward the right shoulder makes the two images come to fusion or approach each other greatly (*C*).

I have given so detailed a description of this method of investigation because it is of the greatest value for differential diagnosis, which I shall consider shortly.

The deviation caused by a trochlear nerve palsy is made up of several components, the vertical one being the most important in point of diag-

nostic value. In typical cases of trochlear nerve palsy the vertical deviation increases in looking down as well as in looking to the sound side, according to the physiologic function of the superior oblique as a depressor muscle. The minimum of vertical deviation or even of binocular single vision is to be found when the patient looks up and to the paretic side. Both the other components of the deviation are of subordinate value for the diagnosis. In many ophthalmologic handbooks an inordinate importance is assigned to the lateral deviation component in palsy of the trochlear nerve. By an isolated contraction of the superior oblique the eye is turned down and out, from which the inference is drawn that it must deviate up and in if that muscle is paralyzed. From this conclusion errors in diagnosis frequently result; palsy of the trochlear nerve is confounded particularly with palsy of the inferior rectus muscle because the paretic eye is turned up and a trifle out instead of in. But this is not decisive because of the frequency of exophoria as a factor in the anomalous position of rest. When a patient with exophoria acquires a trochlear nerve palsy, the exophoria will transform the smaller lateral paretic component from an inward to an outward deviation. Of typical cases of trochlear nerve palsy, at least 25 per cent are without any lateral or with even a divergent deviation.

Only a novice will be induced, on account of the peculiarity just mentioned, to confound palsy of the trochlear nerve with palsy of the inferior rectus muscle, in spite of the fact that the meridian deviations in the two cases are opposite. In a case of palsy of the trochlear nerve there is a torsion outward (disclination); in a case of palsy of the inferior rectus muscle there is a torsion inward (conclination). This difference cannot fail to be noticed on careful investigation, especially if the double vision is examined, not in the old-fashioned manner with a candle for fixation, but with a narrow horizontal object, for instance, the horizontal arm of the tangent scale. The obliquity of the two images of a vertical object like a candle looks quite different (fig. 4) according to whether there is, besides the vertical separation, homonymous or crossed diplopia; these occur, as I have just mentioned, in trochlear nerve palsy. In spite of the same paretic disclination the two images of a candle will converge or diverge upward according to whether there is homonymous or crossed diplopia; in this way a mistake in diagnosis can arise. This mistake is easy to avoid if one uses a horizontal object for testing for vertical and lateral diplopia. In any case of trochlear nerve palsy the paretic disinclination makes the double images converge to the paretic side, whether there is homonymous or crossed diplopia besides vertical diplopia (fig. 4). Figure 5 shows the two images in various parts of the field of fixation in a typical case of trochlear nerve palsy. The patient was fixing the little lamp

in the middle of the tangent scale with a red glass before one eye. By turning the patient's head, while he was ordered to fix the lamp continuously, one made his eyes turn to the left and to the right side, up and down, up and to the left or to the right, and so on. The measurement of the various components of the deviation obtained in this way is not very exact but is sufficient for the diagnosis and as a basis for comparing the results of later examinations with those of the first one.

However, as in cases of abducens nerve palsy, not infrequently trochlear nerve palsy also gradually loses its typical features by the development of a secondary contracture of the inferior oblique muscle, the antagonist of the paretic muscle, while the latter is recovering. In such a case the vertical distance of the two images no longer either

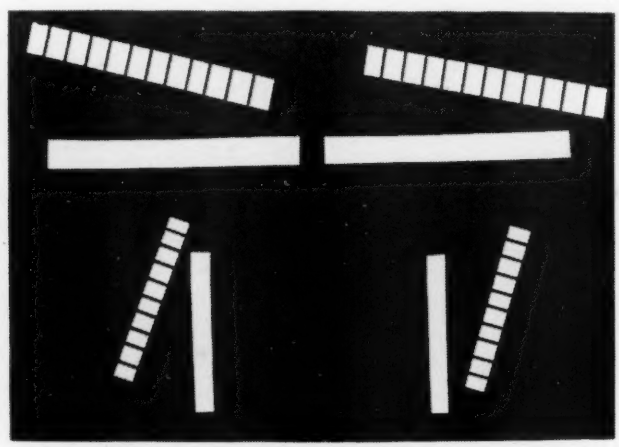

Fig. 4.—Different appearance of the double images of a vertical and of a horizontal object in trochlear nerve palsy according to whether there is, besides the vertical separation, homonymous or crossed diplopia. Though the paretic extorsion is the same in either case, the two images of a vertical object will converge or diverge above, whereas the two images of a horizontal object in those cases will converge always to the paretic side.

increases on looking down or decreases on looking up. Changes in the amount of vertical divergence take place only when the patient is looking from the left to the right; the vertical divergence increases in the direction of the sound side and decreases in the opposite direction, whereas the contrary is noticed with respect to the meridian (rotary) deviation, just as in the first stage of the paresis. Why does the change in the type of the paresis not extend to the influence of the lateral movements on the vertical divergence, so that the deviation becomes concomitant as in abducens nerve palsy? One must consider that the influence of the vertical motor muscles on the position of the eye at a

given moment depends on the angle between the visual line and the muscle planes of those muscles. If the visual line is abducted, the oblique muscles have no influence on the vertical position of the eye, whereas their influence increases the more the visual line is adducted. These conditions are not altered when an originally paretic vertical deviation is at a later stage maintained only by a contracture of the antagonist of the paretic muscle. If one comes across such a case without knowing the first stage of the paresis, it is difficult to decide whether the deviation is to be connected, for instance, with a palsy of the left trochlear nerve or with a paresis of the right superior rectus muscle. In either case the two images have equal vertical deviation, increasing if the patient looks to the right and decreasing if he looks to the left. The small lateral component does not matter, as was said before. Not even the obliquity of the two images is sufficient for the diagnosis.

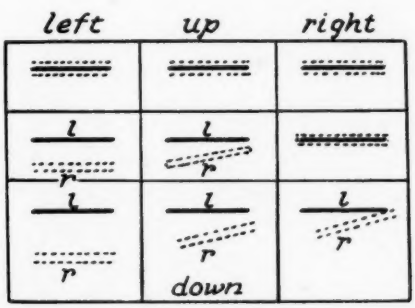

Fig. 5.—Double images (*r* and *l*) of a horizontal object in a typical case of right trochlear nerve palsy in the various parts of the field of fixation.

The novice will be inclined to take the oblique image as belonging to the paretic eye. Indeed, this proves right for the majority of the cases of palsy, but there are many exceptions. In some cases both images appear oblique; in other cases the image which appears upright belongs to the paretic eye and the oblique image to the sound eye. The latter condition occurs not only in long-standing permanent palsy but in fresh palsy if the paretic eye has always had and retains predominance in spite of being paralyzed. In such a case the patient will see a horizontal line correctly, although its image lies not on the horizontal but on an oblique retinal meridian, because of the paretic meridian deviation. So from the kind of obliquity one can conclude only that there is a paretic disclination (extorsion) or conclination (intorsion) of the retinal meridians due to paresis of a muscle rotating the vertical meridian inward or outward; one cannot decide which eye that muscle belongs to.

There is a simple method by means of which one can make sure of the origin of a paresis in which the typical difference in the vertical component on looking up and down is not present. One lets the patient bite on the plate of the little apparatus made for the head-tilting test. He has to observe the two images of the black strip on the cardboard screen. If, for instance, the vertical distance between the two images increases when he tilts his head toward the left shoulder and decreases or disappears when he tilts it the other way, one may conclude that the change in the vertical distance is caused by a disturbed balance of the left oblique muscles, the superior oblique being too weak in relation to its antagonist. If the head-tilting test has no influence on the vertical distance, or if the change does not vary constantly or varies only slightly, one may take it for granted that the muscles of the left eye are intact, but the right superior rectus muscle is too weak in relation to its antagonist. Since this test has proved true in several hundred cases of the palsy under discussion which I saw before the atypical stage developed, I know that it is absolutely reliable, positive results always indicating that the change in the vertical distance between the images is caused by the oblique muscles.

Another method not quite as simple as the one just described for investigating the origin of an atypical vertical interval may be briefly mentioned. Sitting in a dark room with one of his eyes closed, the patient looks through a tube at a faintly glowing electric thread which can be rotated to the left or to the right until it appears to him to be exactly vertical. If at this moment the position of the thread deviates more than 1 or 2 degrees from the real vertical of space, one may infer a corresponding deviation of the vertical meridian of the eye under examination, especially if in the following test with the other eye the patient declares the thread to be vertical as soon as its position really is vertical. This test demands, of course, some intelligence and attention from the patient, but it is useful sometimes if the result of the head-tilting test is negative and one wishes to make sure whether the vertical distance is due to an anomaly of one of the vertical rectus muscles.

In this connection, there may be discussed briefly a peculiar anomaly which is similar to the atypical vertical divergence just described because it produces the impression of excessive functioning of one or both of the inferior oblique muscles (fig. 6). In looking straight forward there is either orthophoria or convergent or divergent strabismus, and sometimes a slight vertical divergence alone or combined with a lateral deviation. When the eyes follow an object moved in the horizontal plane to the left, the right eye will go up considerably, whereas when the patient looks to the left either both visual lines remain on the same

level (fig. 6) or the right eye alone goes up as much as, or more or less than, the left eye had deviated before. One gets the impression that the balance between the oblique muscles is disturbed, the inferior

Fig. 6.—Disturbance of balance between the right oblique muscles: overfunctioning of the inferior oblique. In *A*, both visual lines are parallel in the primary position. In *B*, in levoversion the left eye is going up owing to the abnormal strength of the inferior oblique. In *C*, in dextroversion the visual lines remain parallel because of the normal balance of the left oblique muscles.

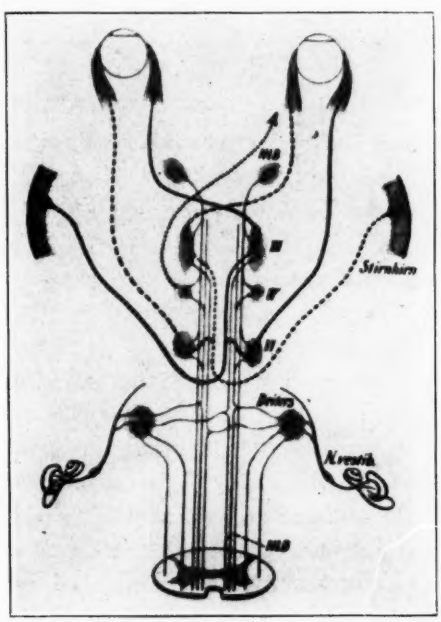

Fig. 7.—Schematic representation (sketched by Villinger and modified by P. A. Jaensch) of the posterior longitudinal bundle (HLB) and its connections with the cortical and vestibular centers and the nuclei governing the ocular movements.

oblique being too strong, so that the more the eye moves inward the more it is raised, because of the unequal functioning of the two oblique muscles. In such cases the field of fixation is enlarged considerably

upward and inward, the direction in which the inferior oblique acts at its best as an elevator, but it is not restricted downward and inward, the direction in which the superior oblique acts at its best as a depressor. The conditions are apparently the same as in long-standing permanent trochlear nerve palsy, in which a contracture of the inferior oblique has developed during the improvement of the paretic superior oblique. The anomaly last described is usually of congenital origin, but in some cases I have seen it arise after Killian's radical operation on the frontal sinus. If in suturing the periosteum the trochlea is not brought back exactly to its former place but remains behind it, the action of the superior oblique will be impaired. But occasionally an overaction of the inferior oblique will result instead, probably because the checking influence of the fascial ligament of this muscle inserted in the trochlea is lessened if the trochlea is pushed behind its normal place. Whether a similar condition is the cause of the congenital overfunctioning of the inferior oblique, as I suppose, is not yet proved. There is a difference between the congenital and the acquired anomaly in that in the former there is no disclination of the vertical meridians of the eye, as is found in every case of the latter, be it a primary anomaly or a secondary contracture of the inferior oblique following trochlear nerve palsy.

OCULOMOTOR NERVE PARALYSIS

Palsy of individual muscles governed by the third nerve is rare in comparison with that of the abducens and of the trochlear nerve. One can dispense with a detailed description, since the signs and symptoms and the principles of the diagnostic analysis are to be derived from what has been said about abducens and trochlear nerve palsies. But paresis of the internal rectus muscle demands special consideration. The internal rectus is the only ocular muscle possessing two functions at the command of the will, one of them obeying the impulse to a parallel movement of the eyes, the other governed by the convergence impulse. Both functions, as well as one of them, may be destroyed, according to where the lesion has taken place. A loss of both functions of the internal rectus muscle without paresis of the third nerve is extremely rare, for it points to a lesion of the nucleus or the roots of the nerve supplying the internal rectus muscle, neither of which can easily be damaged without involving other ganglion cells or other roots of the third nerve. Loss of convergence alone is frequent, owing either to functional or to organic disturbance. Loss of adduction in looking sideways while convergence is intact is less often met with.

In the majority of these cases there is loss of conjugate or lateral movement; that is, the associated external rectus of one eye and internal rectus of the other are paralyzed. Such cases are to be discussed

later. The loss of adduction only in conjugate parallel movements, while adduction is normal or less impaired in convergence, is very interesting and important on account of its value in topical diagnosis. Such a condition can be caused only by a lesion of the posterior longitudinal bundle between the sixth and the third nucleus (ophthalmoplegia internuclearis anterior). This lesion, taking place hard by the third nucleus,

Fig. 8.—Internuclear paralysis of both internal recti due to a lesion of the posterior longitudinal bundle. There is complete loss of adduction in parallel movements to the right (*A*) and to the left (*B*), whereas the maximum convergence is obtainable (*C*).

Fig. 9.—One-sided paralysis of the elevator muscles caused by a supranuclear lesion. In *A*, the left eye is fixing and the right eye is deviated downward. In *B*, on looking up the right visual line cannot pass the horizontal plane. In *C*, the normal function of the right elevator muscles in Bell's phenomenon proves the integrity of the nuclei and roots of the nerves supplying the right elevator muscles.

may cause loss of the adduction faculty of either eye in parallel movements, whereas convergence remains intact (fig. 8).

Besides the internal recti there is only one pair of muscles one-sided paralysis of which can be attributed positively to a supranuclear lesion. Figure 9 shows photographs of a patient with apparently total paralysis

of the right elevator muscles (*A*). The right eye is turned far down, while the left looks straight forward (*B*). The maximum impulse to look up raises the right visual line only to the horizontal position (*B*). But if the patient is asked to close the eyes and the right lids are pulled open, the right eye can be seen to move up perfectly (*C*). The integrity of the right elevator muscles in Bell's phenomenon proves that the nuclei of the nerves supplying the right elevator muscles only are intact; their connections with the cortical centers are destroyed. The lesion must be localized close to the nuclei, below the point where the pathway descending from the cortical center for the elevation of the eyes bifurcates into the branches which go to both the third nuclei. One-sided elevator paralysis of supranuclear origin occurs extremely seldom. I have observed it only three times.

I shall omit discussion of the well known symptoms of typical oculomotor palsy and shall merely point out some of its peculiarities. First,

Fig. 10.—*A* shows total right oculomotor paralysis. In *B*, the right lid fissure opens automatically when the paralyzed internal rectus gets an innervation. In *C*, the patient is able to overcome the paralytic divergence by a strong convergence effort which causes at the same time narrowing of the right pupil and lifting of the right upper lid.

it is striking what extreme deviations many patients are able to overcome by means of a strong convergence impulse in spite of a totally paralyzed third nerve. As a secondary result of the effort, there is to be seen in the photographs in figure 10 a contraction of the right pupil (*C*), which is dilated as long as the paretic deviation is uninfluenced (*B*). Of course, there will also be displayed a spasm of the ciliary muscle produced by that effort. Provided the patient is not too old, he becomes myopic while he is transforming the paretic divergence into parallelism of the visual axes. But these patients learn quickly to relax the ciliary muscles, so that the myopia occurs only as long as the voluntary convergence impulse is being sent to the eyes; it vanishes as soon as binocular single vision is obtained and is sustained no longer by voluntary effort but by the fusion tendency. As the photographs show, the

ptosis is also removed by the impulse to overcome the deviation, although the patient is not able to lift the upper lid when the eye is deviated (*A*).

If the paralyzed eye is constantly fixing because of ametropia or amblyopia of the other eye, the secondary deviation can take really grotesque forms. The photographs in figure 11 illustrate this.

Exophthalmos due to relaxation of the rectus muscles in oculomotor palsy varies considerably in degree. In cases with rather high protrusion there is sometimes a striking retraction movement connected with the abduction of the paralyzed eye. That this symptom is due to the exophthalmos follows from the fact that both these signs disappear at the same time.

RETRACTION MOVEMENTS

A rare phenomenon, called "nystagmus retractorius" by Körber,[5] who first described it, may be mentioned because of its topical diagnostic

Fig. 11.—*A* shows total bilateral oculomotor paralysis with the right eye fixing. In *B*, the left cornea is hidden behind the external angle of the lid fissure as a consequence of the enormous contracture of the left external rectus. In *C*, the left cornea is revealed only by a strong dextroversion impulse.

value. It has been observed only in cases of grave injury of the nuclear region between the third and the fourth ventricle, either diagnosed by the characteristic paretic signs and symptoms of indubitable nuclear origin or proved by autopsy. Elschnig's [6] case was caused by a cysticercus vesicle in the third ventricle; other cases have been due to tumors of this region. Besides paresis of a few or many muscles of both eyes, the most striking sign is a retraction of one or both eyes following every impulse sent to the eye muscles. Elschnig has explained this

5. Körber, H.: Ueber drei Fälle von Retraktionsbewegung des Bulbus, Ophth. Klin. **7**:65, 1903.

6. Elschnig: Nystagmus retractorius, ein cerebrales Herdsymptom, Med. Klin. **9**:8, 1913.

phenomenon as follows: The compression of the whole nuclear region and all its connections, especially the posterior longitudinal bundle, causes disturbance of such a kind that every motor innervation not only excites the ganglion cells which are responsible for the intended movement but spreads over the whole nuclear region, thus bringing about the simultaneous contraction of all the external eye muscles and the consequent retraction movement.

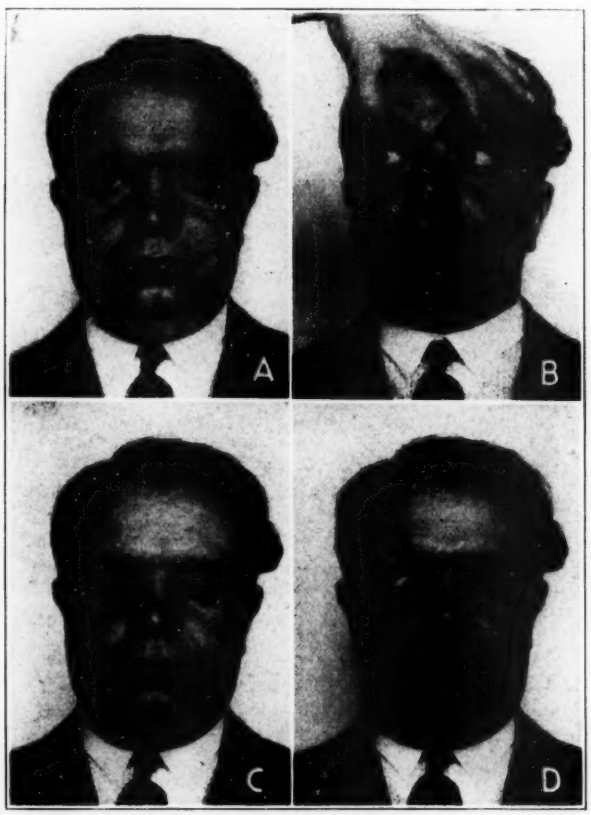

Fig. 12.—Pseudo-Graefe phenomenon as a residue of right oculomotor paralysis. *A* shows that in the primary position slight anisocoria is the only sign of the previous paralysis. *B* shows considerable dilatation of the right pupil in dextroversion. *C* shows equal position of the upper lids in looking down and to the right. *D* shows striking retraction of the right upper lid (pseudo-Graefe phenomenon) and narrowing of the right pupil in looking down and to the left.

PSEUDO-GRAEFE PHENOMENON

Occasionally another peculiar symptom develops in patients partially recovering from oculomotor paralysis. It may be demonstrated by the following examples:

1. A man, 30 years old, acquired paralysis of the right third nerve by fracture of the base of the skull. When I saw him three months after the accident I found, as the only residues of the paralysis, that the right pupil was moderately dilated, the light reaction was hardly obtainable, the convergence reaction was nearly normal and there was slight paresis of the superior rectus muscle (fig. 12). In the primary position the eyes were parallel; the upper lids were equally high (*A*). In looking down and to the right both the upper lids accompanied the eyes normally (*C*), whereas in looking down and to the left the right upper lid was retracted and the right pupil narrowed, so that the anisocoria disappeared.

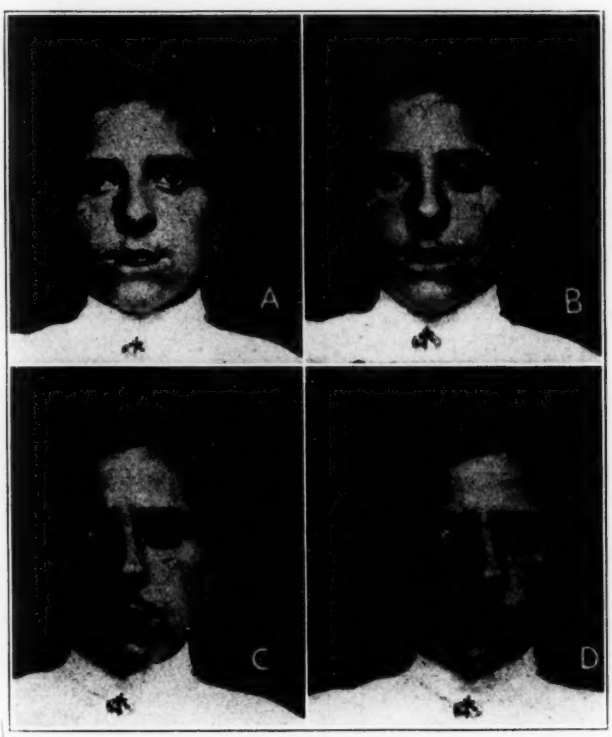

Fig. 13.—In *A*, in the primary position dilatation of the right pupil is the only sign of the previous right oculomotor paralysis. In *B* and *C*, the right upper lid remains unmoved when the impulse to look down or down and to the right is given. In *D*, there is striking retraction of the right upper lid while the patient is looking down and to the left.

2. Nearly the same features were presented by a woman 39 years old six months after a fracture of the base of the skull which had produced total paralysis of the third and seventh nerves. She showed: no ptosis in the primary position of the eyes; normal synergism of the eyes and upper lids in looking down and to the right, and considerable retraction of the right upper lid in looking down and to the left. At the same time the dilated right pupil narrowed; it gave no reaction to light and an imperfect reaction in convergence. I saw the patient again five years later. The only change I found was an increase of the retraction of the right upper lid in looking down and to the left.

3. A woman, 28 years of age, had total right oculomotor paralysis, the origin of which was not determined. Five months later she presented the signs demonstrated by the photographs in figure 13. This case differs from the previous ones in that the upper lid did not go down at all either in looking down or in looking down and to the right. Probably this difference was due to the fact that the depressor muscles had recovered less in this case than in the others. A true retraction of the right upper lid took place only in looking down and to the left. The right pupil behaved exactly as in the other cases.

4. A student had total paralysis of the left third nerve as a result of a fracture of the base of the skull. The most interesting sign of the paralysis in its first

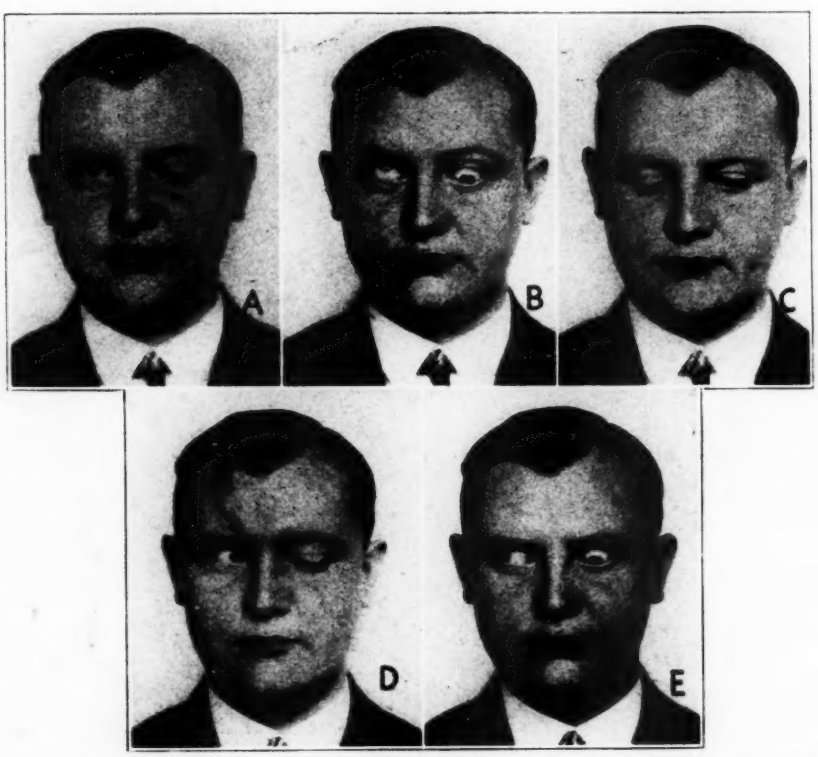

Fig. 14.—*A* shows left oculomotor paralysis, with the left upper lid completely relaxed. In *B*, a maximum contracture of the left superior oblique prevents the left eye from reacting to the elevation impulse, the left upper lid being raised considerably. In *C*, maximum innervation of the depressor muscles causes a slight lifting of the upper lid. *D* shows the levoversion impulse, with complete relaxation of the left upper lid. *E* shows the dextroversion impulse, with maximum retraction of the left upper lid.

stage was the enormous contracture of both the left depressor muscles, chiefly of the superior oblique, which was especially impressive when the patient was looking up (fig. 14). The left eye appeared stationary, while the right eye was looking straight forward, to the right, down or up. Only in looking to the left was there a moderate abduction of the left eye with the same degree of depression

(*C*). The patient was under my observation for more than three years. Not until fifteen months after the fracture was the first change noticed. In the left upper lid, which hitherto had been completely relaxed, some folds appeared and while looking straight forward the patient was able to lift the lid 3 mm. by a maximum effort. On looking to the left, the lid became completely relaxed (*D*) and could not be raised at all, whereas when a strong impulse for looking down was given it rose a few millimeters automatically; this retraction was greater when the right eye looked up (*B*); a maximum retraction of the left upper lid was found in connection with the impulse to look to the right (*E*).

Fig. 15.—*A* shows total left ophthalmoplegia. In *B*, the elevation impulse does not influence the left upper lid. *C* shows the dextroversion impulse, with slight lifting of the left upper lid. In *D*, the left upper lid goes up while the patient is looking down (head thrown back).

5. A woman, 35 years old, whose photographs are shown in figure 15, had basal syphilis, which had caused atrophy of the left optic nerve, paralysis of the left fifth nerve and total left ophthalmoplegia. The left eye was immovable. The left upper lid could not be raised by the strongest effort; it remained relaxed even when the patient looked up (*B*), but when she looked to the right it rose automatically a little (*C*), and in looking down it rose still more (*D*), concurrently with the arrival of the normal right upper lid at its lowest position.

I have seen many patients displaying the so-called pseudo-Graefe phenomenon. E. Fuchs explained the phenomenon as follows: Because the third nucleus is injured or in consequence of a retrograde degeneration has become atrophic, the nervous stimulation sent to a part of the third nucleus overlaps the neighboring parts, so that the latter are stimulated and unintended movements are obtained. I cannot agree with this theory with regard to the patients who displayed the retraction movement of the upper lid in spite of the fact that all the external eye muscles had lost their function completely and the levator palpebrae reacted only automatically to impulses sent to certain other muscles, especially the depressors, which have no physiologic (functional) connections with the levator palpebrae. Further, in nearly all of my patients showing the phenomenon under discussion the paralysis of the third nerve must have been located at the basis cranii. Either there had been a basal fracture or the symptoms additional to the third nerve paralysis necessitated that localization. I cannot imagine a complete retrograde nuclear degeneration, as Fuchs supposed, in which only the nucleus innervating the levator muscle is spared and even this nucleus is not capable of being voluntarily stimulated but responds only to impulses sent to some other atrophic parts of the third nucleus.

I think that the pseudo-Graefe sign can be explained in a less far-fetched manner. Suppose that the continuity of the third nerve is interrupted by a trauma or a tumor. In the course of healing, some of the nerve fibers which proceed from the central part of the trunk of the third nerve do not find their original sheaths in the peripheral part of the nerve but go astray, so that they arrive at muscles to which they do not belong. For instance, the fibers from the nucleus intended for the internal rectus arrive not at this muscle but at the levator of the upper lid, so that the impulse for adduction produces lifting of the upper lid, even if it cannot be lifted by a direct innervation effort because the fibers coming from the levator nucleus have gone astray. In some cases a part of the nerve fibers intended for the levator arrive at this muscle together with fibers of a different origin, so that there is no ptosis but an abnormal retraction of the upper lid as soon as an impulse is sent to certain other eye muscles. It seems that the nerve fibers in the course of healing prefer certain "routes" for growing in the wrong sheaths, so that in the majority of cases the impulse to look down and in produces the strongest contraction of the upper lid levator.

Some cases have been described which presented a similar retraction of the upper lid if an impulse was sent to the paralyzed abducens nerve of the same side, whereas the antagonistic innervation made the upper lid go down. As far as I could make out, the abducens nerve paralysis and the upper lid phenomenon in those cases were of congenital origin,

like another upper lid phenomenon known as jaw winking, an example of which is presented by the photographs in figure 16.

CYCLIC OCULOMOTOR NERVE PARALYSIS

Omitting mention of the rare and less important upper lid phenomena I shall, in conclusion, say a few words about the particularly interesting phenomenon called cyclic oculomotor palsy (Axenfeld), the principal characteristic of which is the automatic alternation of spastic and paralytic conditions of the paretic eye, as the photographs of one of my patients (fig. 17) will show.

The patient was born, as his mother told, with normal eyes. When he was 1 year old the right upper lid gradually drooped. In 1920 he was brought to Uhthoff's clinic, where right oculomotor paralysis and right abducens nerve paresis were noted and an operation was performed to relieve the ptosis. I saw the boy eight years later and noted the automatic alternating spastic and paralytic condi-

Fig. 16.—Jaw-winking phenomenon (sign of Marcus Gunn). *A* shows left congenital ptosis and paresis of the elevator muscles. In *B*, opening of the mouth causes raising of the left upper lid. *C* shows striking retraction of the left upper lid while the jaw is moved to the right. *D* shows relaxation of the left upper lid while the jaw is moved to the left.

tions. In the latter phase there was a nearly complete ptosis in spite of the previous operation (*A*). The right pupil was maximally dilated and rigid (*B*). When the patient was left to himself, one could observe after a fraction of a minute a little twitching arising in the paralyzed upper lid, gradually becoming quicker and livelier and finishing with a complete opening of the lid fissure. At this moment the right pupil, which before had been dilated, would narrow more than the normal left pupil, as the photographs show (*C*). After ten or twenty seconds the right upper lid would slowly go down, and at the same time the right pupil would dilate again. This cycle repeated itself at irregular, but mostly very short, intervals all day and all night, provided the patient was left to himself and did not make voluntary eye movements of great extent. The influence of certain voluntary movements is shown by the subsequent photographs. When the boy was ordered to look to the left during a spastic phase, the automatic alternation would cease and the spastic phase would remain as long as the patient kept the same innervation (*D*). If the order was given during a paralytic phase, this phase would be interrupted after a few seconds and replaced by the spastic phase. The contrary effect was obtained

by the antagonistic voluntary innervation. As soon as this impulse was given the right pupil would dilate and the upper lid would droop (*E*), the condition persisting as long as the boy looked to the right. The last photographs show the position of the right upper lid in looking down during a paralytic (*F*) and during a spastic phase (*G*). These give an impression very similar to that of the pseudo-Graefe phenomenon.

When I collected the reports of cases of so-called cyclic oculomotor paralysis, I found altogether thirty-two; having seen ten cases myself, I am convinced that the condition is not so rare as the small number of

Fig. 17.—Cyclic right oculomotor palsy. *A* and *B* show the paralytic phase: complete ptosis and the right pupil dilated ad maximum. *C* shows the spastic phase: the right lid fissure opened and the right pupil narrowed ad maximum. In *D*, the levoversion impulse elicits the spastic phase. In *E*, the dextroversion impulse elicits the paralytic phase. *F* shows equal position of the upper lids in looking down during a paralytic phase. *G* shows the pseudo-Graefe phenomenon in looking down during a spastic phase.

published cases makes one suppose. There are rudimentary forms of the phenomenon, of which I have just given a rather fully developed example. Only the pupil of the paralyzed eye has shown the cyclic type of paralysis in all the cases observed by me and reported by others. In one third of the cases the upper lid does not participate in the cyclic

phenomena. Cases in which the internal rectus muscle shares in the cycle are fewer still, and it is exceptional to find the inferior rectus actively involved. The elevator muscles never participate in the alternating spasms and relaxations. So it is easily understood that mild cases, presenting perhaps only the phenomenon of automatically alternating dilatation and contraction of the apparently paralyzed pupil, are overlooked. Some of them have been reported as cases of third nerve paralysis showing a peculiar pupil-phenomenon. In about 50 per cent of the cases the phenomenon is not congenital but acquired in early childhood; in one case it did not appear until the seventeenth year of life. In the majority of cases the paralyzed eye is highly amblyopic or ametropic. About the localization and the origin of the lesion causing the phenomenon, only theories have been advanced. In my publication in von Graefe's *Archiv für Ophthalmologie*[7] I discussed the problem at length and explained why, in my opinion, the lesion must be localized in the region of the third nucleus. Varying vasomotor influences probably play a part in bringing about the apparently automatic alternation of spastic and paralytic phases. The interesting influence of voluntary movement impulses in the course of the phenomenon I have tried to explain as follows: A part of the oculomotor nucleus, which has retained but a slight degree of function, reacts only if it is abundantly provided with blood. This is obtained when, by sending an impulse to the oculomotor nucleus, the blood vessels in this region are made to dilate; after a short interval the spastic phase arises. On the other hand, an antagonistic impulse going to the abducens nerve of the paralyzed eye causes an inhibition of the oculomotor excitation by the contraction of its blood vessels; the paralytic phase results. The reasons for my theory are given in detail in the publication just mentioned.

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7. Bielschowsky, A.: Ueber die Oculomotoriuslähmungen mit cyclischem Wechsel von Krampf- und Erschlaffungszuständen am gelähmten Auge, Arch. f. Ophth. **121**:659, 1929.

TESTING THE EYES OF SCHOOL CHILDREN

A NEW EYE TEST CABINET

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Testing eyes in the schools is unlike testing them anywhere else. It is a screening process done in an attempt to sift the children with good eyes from those with bad eyes and to have the latter placed under the care and attention of a physician. The peculiar problems confronted in testing the visual acuity of pupils in schools are not readily understood by many physicians. Even the ophthalmologists are frequently unaware of the obstacles encountered in examining the vision of hundreds or thousands of children in a given school or school system. The matter of charts, their illumination, the illumination of the room, the correction for the angle of vision, the portability of the charts, the children themselves, the memorizing of the charts, the examiners, the procedure in testing, the scoring and the follow-up study are so varied, not only in each school system but in each school building, that the nurses or teachers who usually do the testing, and who also have many other duties to perform, cannot become as proficient as the nurse or assistant in the ophthalmologist's office who does nothing but ocular work.

First will be mentioned the various difficulties which have been encountered in testing the visual acuity of the children in the Albany school system and then will be explained the chart or cabinet that has been devised to overcome these problems.

DIFFICULTIES IN TESTING

Testing the vision of the children in a school system is a wholesale job. Hundreds of thousands of children must be tested soon after school begins, so that their vision may be corrected at the beginning of the school year. A number of children are frequently brought in at one time. It is common knowledge that charts or portions of charts are often memorized. The ideal chart should do away with this.

It must be remembered that dark classrooms, offices and even corridors are frequently the only available places in some school buildings for testing the eyes. Illumination is varied in the different schools.

Not uncommonly the overhanging lights or side lights glare into the eyes and distract from the test. The ideal test chart should obviate the use of these obnoxious lights. There should be some standard illumination, just the number of foot-candles of light necessary for visual comfort. From 15 to 20 foot-candles of light on the eye chart as sometimes recommended may give an uncomfortable glare. The amount of light on the charts depends on the make-up of the chart itself as well as on the details of the light source. There are many kinds of cards and cabinets used for testing the eyes. Some have colored distracting lines, some have clear white backgrounds and others have darker backgrounds. The Snellen type of chart on which the lines are marked off in feet seems to be the best. Every type of reflected light is used with the cards, which are usually placed in any available space. Often there is no outlet for proper electrical fixtures, and as a result the light on the test card is poor. Cabinets in general are better than the test cards, as the illumination is more definite.

To be really scientific, a chart should be correct for the angle of vision: horizontal, vertical or oblique. With the ordinary test card there is a 2 foot (60.9 cm.) vertical range of symbols. Each line tested should be placed on a direct line with the child's vision, and the line of the chart should be on a level with the child's eyes, i. e., raised for the taller child and lowered for the shorter child.

The chart should be portable so that it may be used in the health or other available room and also in any classroom. In the larger school districts testing the vision of pupils may be made a classroom activity. In the smaller school districts the portable chart could be taken from one school to another.

The procedures for testing the eyes and scoring of the results as well as for the follow-up study are variable, depending on the particular technic of the teachers, nurses or physicians conducting the tests. It seems that many wasted steps are taken in testing children's eyes. The child is brought in; the chart is explained; the child is placed at the 20 foot line and is told how to cover his eye; the examiner points to the letters or figures on the chart; the results are then written on the pupil's health record. This procedure is repeated for the other eye. There is much going back and forth during these procedures. It would be ideal and would save considerable time to have an arrangement whereby the examiner could remain at her desk from the beginning of the test until the time the record is completed. Instructions concerning procedure, scoring and follow-up study might be placed on the chart in some standard fashion, visible to the examiner only. In this manner information concerning the testing of the eyes and the particulars for each line tested could be before the examiner constantly.

THE NEW EYE TEST CABINET

To overcome these various problems and difficulties encountered in testing the eyes of children in the Albany school system an eye test cabinet has been devised which has proved ideal for many reasons (fig. 1). It has a smooth, even, diffuse illumination and is lighted by incandescent filaments from behind. The varied conditions of illumination under which eyes are tested in the schools undoubtedly affect the results of many tests. This cabinet makes it unnecessary to have distracting and glaring overhanging or side lights. The symbols can be seen with comfort and ease. It is not necessary to have a dark-

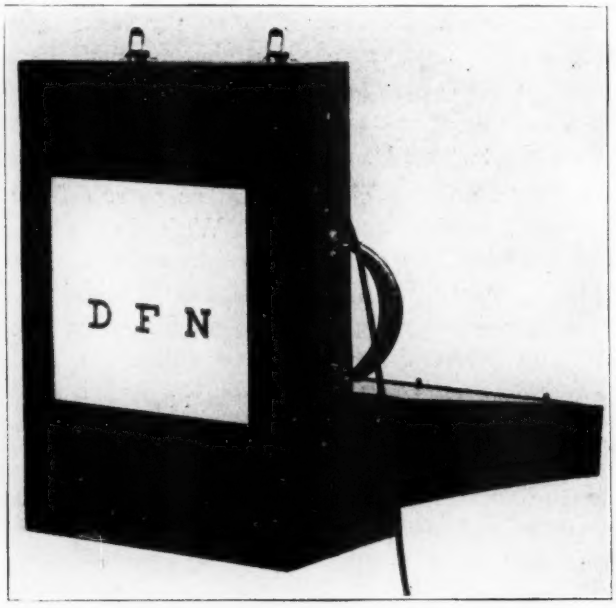

Fig. 1.—The new eye test cabinet.

room effect. The rear of the cabinet is illuminated and when opened forms a desk top which permits the examiner to make notations on the health records, even though the room is in darkness (fig. 2).

One line of the chart is shown at a time; the rest are entirely hidden. There is a blank space between each line which prevents memorizing. Each line of characters may be brought to the direct level of each child's line of vision. This prevents an error in the angle of vision. The cabinet placed on a table or desk is at a proper height for small children. Taller children or adults may be tested in a sitting posture. Unhandy, cumbersome and intrinsic means for placing the chart at proper levels for vision are thus obviated.

There is synchronous text to correspond with each line of vision tested (fig. 2). The text, which conforms with methods advocated by

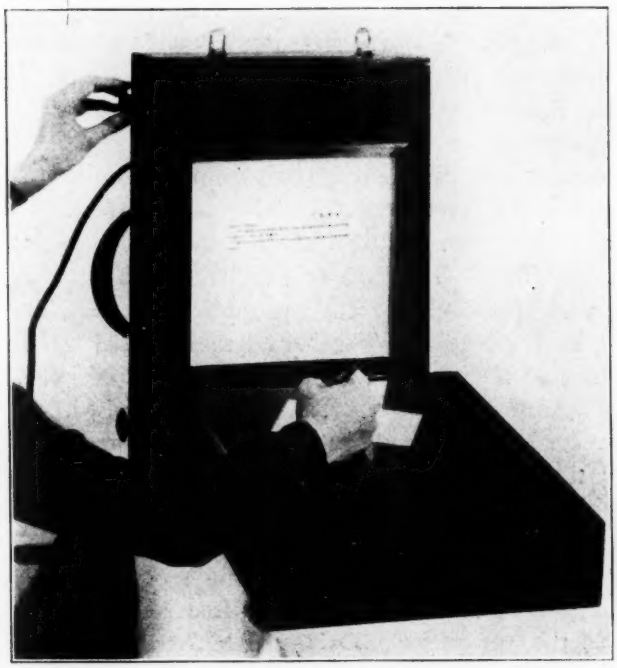

Fig. 2.—The cabinet open, showing how it may be used as a desk top.

Fig. 3.—The illustration displayed on the cabinet shows the proper way for the subject to cover one eye while the other is being tested.

the New York State Department of Education,[1] is visible to the examiner, while the chart is visible to the person being examined.

There are illustrations on the chart showing the proper way to cover one eye while the other is being tested (fig. 3).

The examiner manipulates the chart from beginning to end without rising, regardless of the number of children examined. The examiner, in addition to being confronted with the text, may easily point to any symbol on the chart. The children are tested in rapid succession in accordance with the monitor system prescribed by the New York State Department of Education.[1] The varied means used by the examiners for procedure, scoring and follow-up study are eliminated. A definite standard to insure uniform results is thus established.

Actual records for the past two school years, involving over 30,000 tests, show that for pupils examined in all grades the average time per child with this test cabinet, including recording, was one minute and fifteen seconds per pupil. Formerly, using the Snellen chart under varied conditions, the time for testing and recording was from three to eight minutes. Further reduction in time is secured by having the children read the lines nearer 20/20 rather than starting at the 20/200 line. For example, the testing might begin at 20/50 or at whatever line the previous test happened to end. Some examiners, however, prefer to start at the top of the chart in order to give the eyes plenty of time to accommodate themselves.

When closed, the cabinet is readily portable.

A survey among nearly all of the local ophthalmologists disclosed the fact that over 90 per cent of the pupils referred to them for vision require care and attention.

This eye test cabinet affords good illumination, provides for the proper angle of vision, is readily portable, prevents memorizing, lessens testing time and simplifies and standardizes procedure in testing, scoring and follow-up study.

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1. Pratt, Emily A.: Specialist for Eyes and Ears, in The Eyes and Ears of School Children, New York University Health Bull. no. 2, 1924.

THE PHYSIOLOGIC OPTICS OF RETINOSCOPY

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Following the introduction of retinoscopy by Bowman in 1859 and its use in 1873 by Cuignet and his pupils, attempts were made to explain the underlying physiologic optics. Cuignet's [1] analysis was soon followed by that of Parent,[2] who was credited by Tscherning [3] with first giving the correct explanation. The theory of Leroy,[4] which is essentially in agreement with that of Parent, is widely accepted. Briefly stated, the theory is that the observer sees as light only that part of the pupil which is sending light to it. Essentially the explanations of these workers are to be found in the modern textbooks of physiology and ophthalmology.

In teaching physiologic optics and retinoscopy to medical students I have felt that the available analyses were not entirely adequate. Somewhere along the way it becomes impossible to proceed rigorously with the explanation. Particularly is this true in attempting to analyze the phenomenon of the "reversal point." Some authors, in discussing the optics of retinoscopy, omit a real analysis of this specific situation (Alger,[5] de Schweinitz [6] and Howell [7]), whereas others analyze it incorrectly. Burnett,[8] for instance, stated that the image at the observer's nodal point of the source of light on the subject's retina "becomes itself an illuminated object which sends out rays in all directions, and . . . the fundus of the observer will be fully illuminated." In the "American Encyclopedia of Ophthalmology" [9] the same statement is made. That

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From the Department of Physiology, University of Chicago.

1. Cuignet, cited by Tscherning.[3]

2. Parent, H.: Rec. d'opht. **3**:229, 1881.

3. Tscherning: Physiologic Optics, Dioptrics of the Eye, Functions of the Retina, Ocular Movements, and Binocular Vision, translated by Carl Weiland, Philadelphia, The Keystone Press, 1900.

4. Leroy, C. J. A.: Rev. gén. d'opht. **6**:290, 1887.

5. Alger, E. M.: Refraction and Motility of the Eye, Philadelphia, F. A. Davis Company, 1910.

6. de Schweinitz, G. F.: Diseases of the Eye, Philadelphia, W. B. Saunders Company, 1921.

7. Howell, J. A.: Text-Book of Physiology for Medical Students and Physicians, Philadelphia, W. B. Saunders Company, 1933.

8. Burnett, S. M.: The Principles of Refraction in the Human Eye Based on the Laws of Conjugate Foci, Philadelphia, The Keystone Press, 1904.

9. Wood, C. A.: American Encyclopedia and Dictionary of Ophthalmology, Chicago, The Cleveland Press, 1918, vol. 13, p. 9924.

this cannot be so may be verified by any ophthalmologist, who will recall that his retina is not diffusely lighted but is lighted only over an area definitely bounded by the image of the observed pupil. Again, Thorington [10] stated that "if myopia should be just 1 diopter, the emergent rays would focus on the observer's retina at 1 meter." Howell [7] also made essentially the same statement. But this is not the case, for one does not get a sharp image of any detail of the observed retina at the point of reversal. As a matter of fact, at no time during a retinoscopic examination is retinal detail seen; only a diffuse light from a source "out of focus" is observed.

Difficulties of this sort have led me to make a somewhat different approach to the problem. An attempt is made to analyze the course of the light rays involved from the subject's retina to the observer's retina —to explain what the observer sees on the common sense basis of exactly what is happening on his retina, that is, exactly which areas are lighted and which are dark. All the writers previously mentioned have carried their analyses to the point where the emergent rays reach the cornea or the pupil of the observer. Others, for instance Parsons,[11] have diagrammed the course of the rays to the retina in a manner not in accord with optical principles. Further, according to this diagram, a definite movement of shadow should be observed at the point of reversal before the subject's pupil appears completely dark, which is not what the ophthalmologist observes.

In order to make the proposed analysis, it is necessary to bear in mind that the observer's eyes accommodate themselves for the plane of the subject's pupil and maintain this accommodation during the entire test. The ophthalmologist recalls that throughout the examination he sees the margins of the pupil clearly. Illustrations in textbooks of what the observer sees show the pupil always in sharp focus and the retinal source of light blurred. This point has been duly emphasized by Tscherning,[3] Parsons,[11] Duke-Elder [12] and others, but is not always appreciated, as when Thorington [10] stated that the observer's retina and the subject's retina (not pupil) are in conjugate foci.

This situation is diagrammed in figure 1 in the conventional manner. On the observer's retina an image *ab* is formed of the pupil *AB* of the subject. That is, at point *a* are focused all rays of light from point *A* of the subject, and at *b*, all rays from *B*. It is, of course,

10. Thorington, J.: Retinoscopy (or Shade Test) in the Determination of Refraction at One Meter Distance from the Plane Mirror, Philadelphia, P. Blakiston's Son & Co., 1911.

11. Parsons, J. H.: Diseases of the Eye, New York, The MacMillan Company, 1930.

12. Duke-Elder, W. S.: Text Book of Ophthalmology, London, Henry Kimpton, 1932, vol. 1.

true that any changes the observer notes in the course of his examination will be due to the changes in the manner in which the area *ab* of his retina is stimulated. Movements of light or shadow will be limited to this area. No other part of the retina is involved. The object, then, is to analyze exactly what occurs on this critical portion of the retina.

The crux of the matter lies in the fact that the observer's eyes remain accommodated for plane *AB* throughout the test. The direct consequence of this is that any ray of light coursing along a path from point *A* to any point between *D* and *E* will reach and stimulate

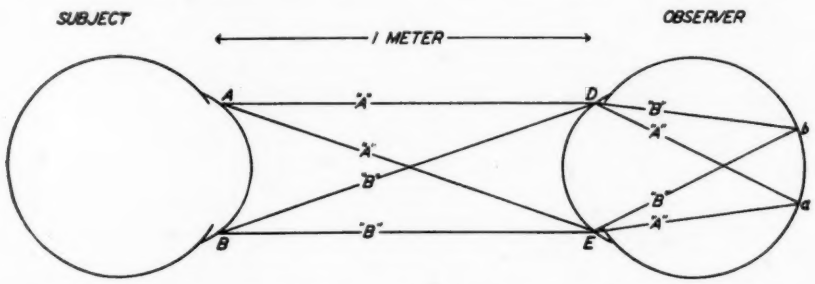

Fig. 1.—Diagram showing the formation of the image of the subject's pupil on the observer's retina.

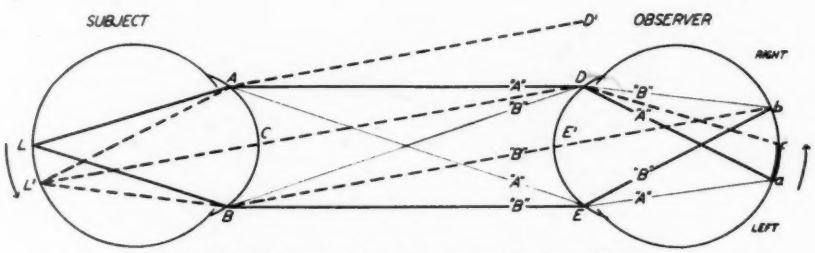

Fig. 2.—Diagram for the emmetropic subject.

the observer's retina at *a*. This holds no matter what the previous history of the light ray has been. For convenience, any such ray will be called an *A* ray in the subsequent analysis and will be labeled so in the diagrams. Similarly, any ray of light, from whatever source, so long as it traverses a path from *B* to any point between *D* and *E*, will strike the observer's retina at *b*. Such rays will be referred to and labeled *B* rays.

Suppose the subject is emmetropic. His retina is made a source of reflected light in the usual manner. Consider this light to be reflected from point *L* and emerging from the subject's eye in parallel bundles, as depicted by the full heavy lines in figure 2. The figure also retains the light lines *AE* and *BD* depicting the course of the rays from

points A and B, for which the observer's eyes continue to accommodate. These now become important reference lines. The emergent parallel rays are bounded by rays AD and BE. It is at once seen that ray AD is an A ray and that it will be refracted to point a. Similarly, ray BE is a B ray and will reach the observer's retina at b. Rays between AD and BE, but parallel to them, all coming from L, will stimulate the observer's retina between points a and b. The entire retinal region ab will consequently be lighted, which is to say that the entire pupil of the subject will appear lighted.

If now the observer rotates his flat mirror to the left, the reflected source of light on the subject's retina will actually move to the left.

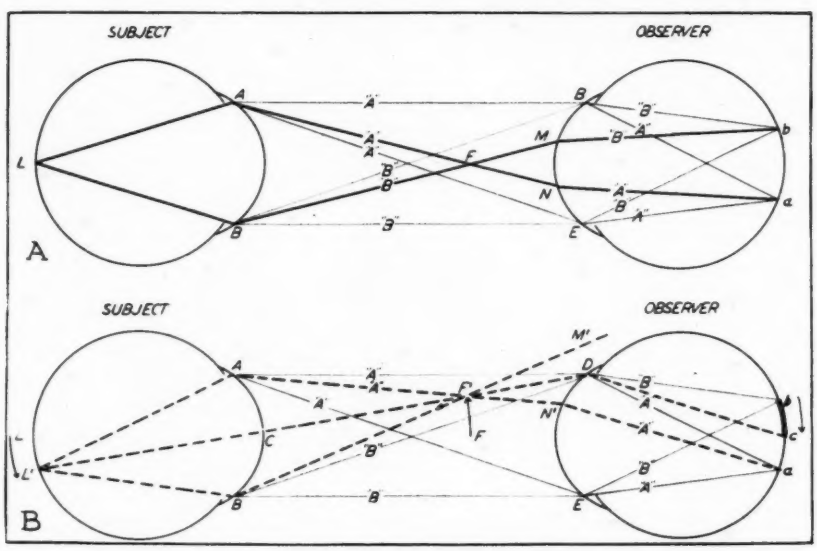

Fig. 3.—Diagram for the eye with myopia of greater than 1 diopter.

L' now becomes the source of reflected light. The emerging bundle of parallel rays (shown in broken lines) are bounded by rays AD' and BE'. Note that while AD' now does not enter the observer's eye, BE' not only does enter but remains a B ray and will reach the observer's retina at b. That ray BE' has come from L' does not alter the fact that its direction coincides with that of a B ray, i. e., with a ray of the pencil BDE. I have shown that all rays of this cone reach the retina at b by virtue of the observer's continued accommodation for plane AB. Ray CD, which barely enters the right extremity of the observer's pupil, is neither an A nor a B ray. Its direction is intermediate between these, and it will reach the observer's retina at some intermediate point, c. The observer's retina will now be lighted between points b and c. Region ac is dark. This movement of the shadow

from *a* to *c* on the observer's retina will, of course, be interpreted as movement of the shadow "across the subject's pupil" in the reverse direction by virtue of the faculty of central projection. I thus have explained why rotation of the flat mirror to the left is seen as movement of the shadow to the left when the subject is emmetropic.

Figure 3 *A* shows the course of rays in a case of myopia of greater than 1 diopter. Rays emerging from *L* converge to *F* and reach the observer's eye as rays *BM* and *AN*, plus all intermediate rays. Note that ray *AN* is an *A* ray and will continue therefore to *a*, and that *BM* is a *B* ray and will arrive at *b*. Again the whole area *ab* is lighted. Causing the reflected source to move to *L'* (figure 3 *B*) shifts *F* to *F'*. Ray *BM'* does not enter the observer's eyes, but ray *AN'*, which does, is still an *A* ray despite the shift and, as before, is refracted to point *a*. Marginal ray *CD* is intermediate between *A* and *B* rays and strikes the retina at *c*, intermediate between *a* and *b*. Region *ab* is now lighted from *a* to *c* and dark from *c* to *b*. In this case the

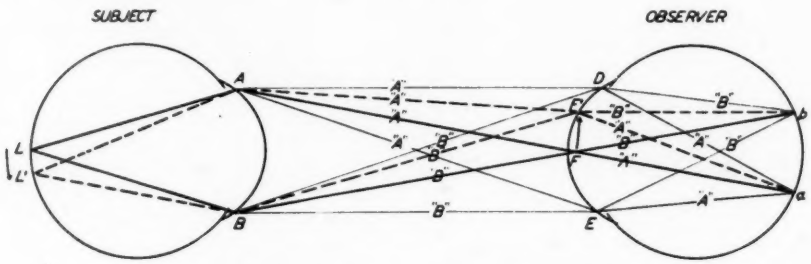

Fig. 4.—Diagram showing the point of reversal.

shaded portion of the retina passes from right to left, exactly the reverse of the direction of movement found when the subject is emmetropic.

This type of analysis is particularly well adapted to explain the most puzzling (and intriguing) phenomenon of the test—the point of reversal. Whereas in figure 2 the movement observed is with the plane mirror and in figure 3 *B* against it, at the point of reversal no movement can be detected until the mirror has been rotated sufficiently to cause the light to disappear regardless of the direction of rotation.

Figure 4 shows what occurs in this situation. Rays reflected from *L* emerge from the subject's eye to converge to point *F*. Ray *AF* is an *A* ray, destined to go to *a*, and ray *BF* is a *B* ray and must reach *b*. Intermediate rays in the cone *AFB* will stimulate the retina at points intermediate between *a* and *b*. If *L* is moved to *L'*, shifting *F* to *F'*, *AF'* is still an *A* ray, and is still refracted to *a*, and *BF'* remains a *B* ray and continues to *b*. The entire area *ab* has remained lighted despite the shift, and the subject's entire pupil remains lighted exactly as before

the shift. It will readily be seen that this will hold no matter to which position *L* is shifted, so long as point *F*, the apex of the cone, falls between points *D* and *E*, the margins of the observer's pupil. When *L* is moved so that *F* falls outside these boundaries, the entire area *ab* will be dark and the subject's pupil will have appeared to become dark, preceded by no detectable movements of light or shadow.

That the reflected source of light on the subject's retina is not really a point source but an area comprising numerous points in no wise vitiates the analysis. To take this fact into account one should merely have to repeat the foregoing constructions for each of the assembled points, which is impracticable to diagram. To consider the source as an area is helpful in the analysis, especially of hyperopia; otherwise, the situation in hyperopia (or in myopia of less than 1 diopter) is similar to that in emmetropia, as shown in figure 2.

It should also be apparent that the analysis is by no means invalidated by the observer's failing to focus sharply for the plane of the subject's pupil. Such a possibility could be illustrated merely by moving the observer's retina forward or backward slightly without altering another line in the diagrams. The only result of such a failure to accommodate would be to cause the margin of the pupil observed to appear blurred. All movements of light and shadow would be the same.

In Duke-Elder's [12] discussion of the optics of retinoscopy, he made use of the principles here outlined in his description of the optical conditions at the point of reversal, after a discussion of retinoscopy in which no other attempt was made to analyze what actually happens on the observer's retina.

I believe this to be a simple and sound analysis of the rather elusive optical and physiologic background of retinoscopy, of interest both to the ophthalmologist who employs the test daily and to the modern, inquisitive medical student, who wants to know how and why.

CHEMISTRY OF THE LENS

VII. GROWTH AND SENESCENCE

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Ophthalmologists have been interested in the relation of growth and senescence of the human lens to ocular diseases because one course of pathologic events would occur in the eye if the lens were to grow continuously in size and another course would take place if the lens were to cease growing and become senile.

The growth of the human lens from birth to death has been assumed to be continuous. The physiologic argument for the continuous enlargement of the lens which has been advanced by Priestly Smith [1] is that the lenticular cells "unlike those of the cuticle have no free surface and are not cast off as they grow" and that "they multiply within a closed capsule, and are laid down layer upon layer in such a way that the older are surrounded by the younger." If it is true, as Priestly Smith said, that the lenticular growth is continuous, then the lens does not behave like most of the tissues of the body but is similar to cuticle, hair, nails, connective tissue and blood cells.

The only published data on the growth of the human lens are those of Priestly Smith. The averages he obtained for one hundred and twenty-eight clear lenses are as follows:

Age, Years	Weight, Mg.	Volume, C. Mm.	Specific Gravity
20 to 29	174	163	1.067
30 to 39	192	177	1.085
40 to 49	204	188	1.085
50 to 59	221	205	1.078
60 to 69	240	225	1.067
70 to 79	245	227	1.079
80 to 90	266	244	1.090

A plotting of his data on the weights of one hundred and twenty-eight clear lenses and the ages of the subjects on coordinate paper shows practically a constant gain in weight of the lens in relation to age. In other words, the growth of the human lens is anomalous. When his results are closely examined certain unusual differences are noted. The average gain in weight of the lens during the eighth decade of life is

From the Wilmer Ophthalmological Institute of the Johns Hopkins Hospital and University.

1. Smith, Priestly: On the Growth of the Crystalline Lens and Capsule, Tr. Ophth. Soc. U. Kingdom **3**:79, 1883.

from one-third to one-fourth as great as in the other decades, and the increase of volume is from one-tenth to one-fifth as great as in the other decades. A similar but less marked difference is shown in the fourth decade. The specific gravity of the various lenses shows great irregular variations. Apparently the values of the weights of the lens as recorded by Priestly Smith are the most consistent of his data. Several unanswerable questions arise in connection with the report of Priestly Smith. 1. How were the lenses removed from the eye? If they were obtained by dissection the weights of the lenses would approach a true weight. If they were "extracted" [1] by expression through an incision of the cornea the lenses would weigh less than they should, as extraction causes a loss of water in the lens. 2. How long after death were the lenses removed? The animal lens slowly increases in weight after death. For these reasons the weight of the human lens in relation to time should be reinvestigated.

The meager data of Jess [2] on the weight of the bovine lens at various ages were used to study the growth of the lens. The curve which was plotted from his data by Krause [3] suggested that the growth of the bovine lens approaches a maximum like most of the other tissues of the body and is not anomalous. The curve showed that the growth of the bovine lens is autocatalytic and of the nature of a monomolecular chemical reaction.

In order to determine whether this fact is true for the growth of the lens of other animals the investigation here reported was carried out.

EXPERIMENTAL DATA

Because growth involves an increase either in size or in differentiation or in both, the measurement of lenticular growth should include both of these factors. Since the measurement of increase in differentiation or of increase in volume presents experimental difficulties the increase of growth in the lens may be most conveniently measured by the increase in weight.

The eyes of pedigreed rabbits were made available through the courtesy of Dr. E. L. Burky. The rabbits were inbred albino and mixed colored stock. The complete breeding history of each rabbit was recorded. The females generally weighed more than the males of corresponding age. All the rabbits ate the same diet. The eyes were excised from the rabbits as soon as possible after death. The lenses with the unbroken capsule were removed by dissection of the eye and weighed immediately in glass-stoppered weighing bottles.

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2. Jess, A.: Beiträge zur Kenntnis der Chemie der normalen und der pathologischen veränderten Linse des Auges, Ztschr. f. Biol. **61**:92, 1913.

3. Krause, A. C.: Chemistry of the Lens: IV. The Nature of the Lenticular Proteins, Am. J. Ophth. **17**:502, 1934.

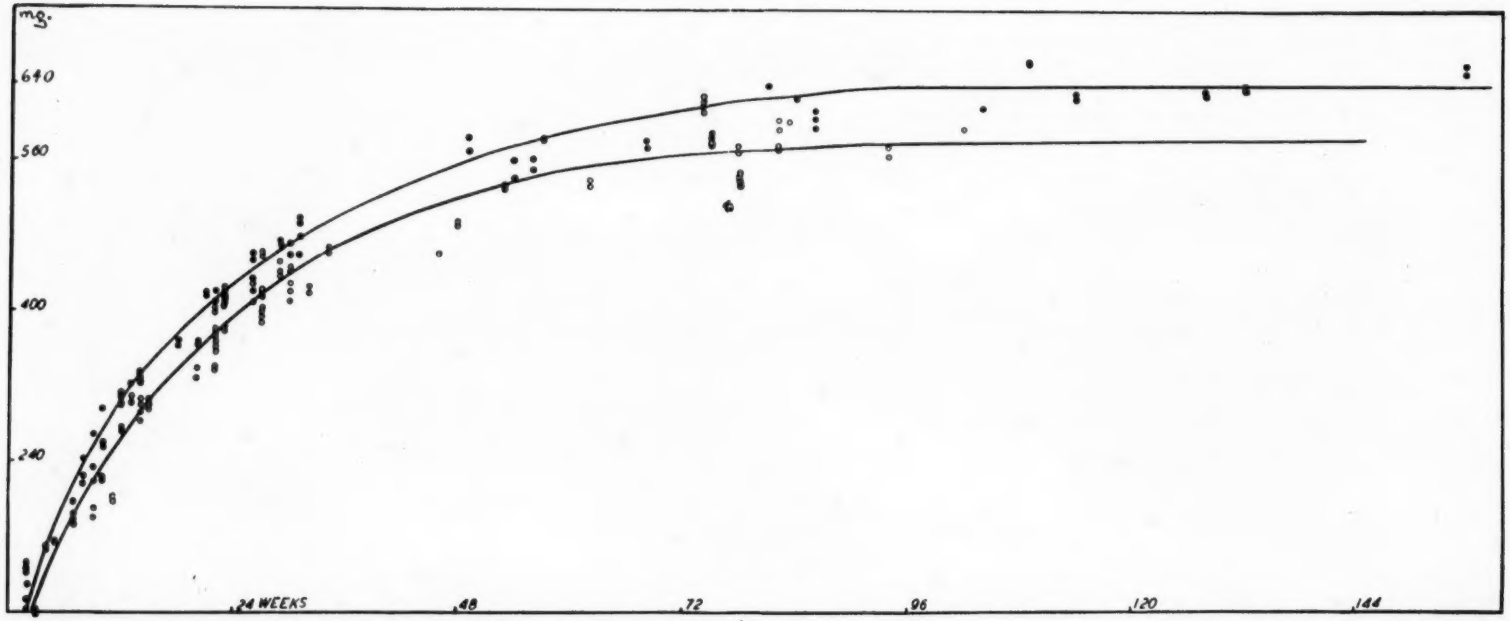
Weight of the Lens of the Rabbit in Relation to Age

Pigmented Female			
		Weight, Mg.	
Animal	Age, Weeks	Right Eye	Left Eye
1	2	83	92
2	2	122	107
3	2	133	123
4	3	75	82
5	4	150	146
6	7	172	177
7	7	185	197
8	8	216	225
9	10	220	...
10	10	258	258
11	10	294	...
12	12	294	297
13	12	...	314
14	13	321	301
15	14	334	329
16	14	318	322
17	15	307	304
18	36	431	425
19	49	584	570
20	53	535	530
21	54	561	542
22	56	552	561
23	57	580	585
24	68	573	582
25	81	...	640
26	84	628	..
27	114	626	631
28	128	633	630
29	132	635	637
30	156	661	652
Pigmented Male			
31	5	155	153
32	8	241	221
33	9	234	..
34	9	...	270
35	10	218	221
36	12	296	302
37	12	274	271
38	18	364	369
39	20	367	369
40	21	416	421
41	22	421	412
42	23	415	419
43	25	460	465
44	26	454	461
45	26	422	432
46	29	475	472
47	30	471	463
48	31	481	459
49	31	495	498
50	75	590	586
51	86	586	591
52	86	594	...
53	104	616	...
54	109	661	666

Albino Female			
		Weight, Mg.	
Animal	Age, Weeks	Right Eye	Left Eye
1	11	208	195
2	13	307	302
3	14	306	299
4	14	282	293
5	22	370	367
6	23	415	419
7	23	342	348
8	27	387	398
9	30	421	410
10	30	443	446
11	32	426	419
12	34	468	462
13	62	532	539
14	75	580	584
15	77	515	511
16	78	575	570
17	82	572	573
18	82	592	603
19	102	594	...
Albino Male			
20	1	62	...
21	9	223	...
22	9	185	191
23	14	370	369
24	15	301	306
25	20	348	330
26	22	340	336
27	22	375	380
28	22	401	402
29	22	361	352
30	22	368	351
31	22.5	389	380
32	23	423	420
33	26	432	411
34	27	419	421
35	27	423	420
36	27	462	460
37	27	402	398
38	29	...	452
39	29	442	439
40	30	...	431
41	46	460	464
42	48	497	490
43	78	550	544
44	78	540	537
45	83	601	...
46	94	572	564

The table gives the age of the animal in weeks and the weights of the lenses of the right and left eyes. The lenses of the male and female rabbits vary within the same small limits. The sex of the animal is not indicated in the chart.

The lenses of the pigmented rabbits were larger than those of the albino rabbits of corresponding age. The weights of the lenses of four different pure breeds of rabbits of various known ages were also determined in this laboratory. These unpublished and published results also show definitely that the growth in weight of the lens is dependent on the breed of the animal; in man it is probably dependent on the racial stock. In general, a large-sized breed of rabbit possesses larger lenses than a small-sized breed.



Weights of the individual lenses in milligrams as plotted against the age of the rabbit in weeks. The upper curve shows the growth of the lens of the pigmented rabbit (black dots); the lower curve shows the growth of the lens of the albino rabbit (circles)

The results of the study of the growth in weight of the lenses of all kinds of rabbits indicate that the lens grows in the autocatalytic manner and that it stops growing when it reaches a maximal weight.

COMMENT

With respect to its growth and senescence the story of the life of the lens should begin with the lens of the prototypic cerebral eye of the vertebrate and end with the human lens. It should include the genetics, comparative anatomy, cytology, embryology, physiology and chemistry of the lens. The history of the lens should also be traced through the genes, the units of inheritance, for it is through them that the inheritance of the lens has been carried through the evolutionary stages of various animals to man. Since the genes of the chromosomes are composed mostly of chromatin, a protamine combined with nucleic acid, it may be said, in the strict sense, that the study of the growth and senescence of the lens should begin with the inheritance of chemical compounds and the inheritance of chemical reactions. Intensive experimentation has shown that the genes producing reactions controlling the characteristics of the eye of the fruit fly, Drosophila, may be quite precisely located in different chromosomes. The shift of genetic chromatin is regarded as a result of translocations of chromatin. However, no such genetic and cytologic research on the relation of the genes to the vertebrate lens has been attempted. The next step forward in this genetic and cytologic study of lenticular growth should be the chemical investigation of the relation of the genes to the lens. Apparently the chemistry involved in this problem of chemical genetics of the lens is beyond the present methods of attack, but the answer will doubtlessly be obtained in the near future.

Another part of the problem of growth and senescence of the lens is a consideration of the chemical factors which determine the relation of the growth of the lenticular cells to other cells. The processes of development in the eye are interdependent and are definitely planned in space and time. The typical lens is formed only if the process of development is initiated and is continued with definite substances under the influence of the chromosomes. The development of the lens is controlled by two factors. The independent factor is shown by the development of the lens in the eye when the cells of the primordium of the whole eye in the vertebrate embryo are transplanted. The dependent factor is illustrated by a lack of development of the lens when the skin of the head which is to form the lens of the eye is isolated, showing that the presence of an eye is necessary for the development of the lens. Ultimately this will be a problem for chemical research on the growth of the eye.

The growth of the prenatal lens has been followed histologically but not chemically. After the lens plate is formed in the embryo the lens develops rapidly in size until birth. In the prenatal cells the amount of nuclear material decreases and the amount of cytoplasm increases. In the postnatal lens the cells are apparently limited in number, and under normal conditions practically no natural proliferation of the cells occurs, although they may grow in length to form lens fibers.

The gross growth of the lens, which is due to an increase in the number of the cells and to prolongation of the fibers, is a complex problem. The early embryonic lenticular cell grows to a definite size and then divides into two cells. Why do cells divide, and why do they grow to a definite size? The theory is that the cell grows when it is in unstable equilibrium with the medium surrounding it, and the increase in size is an expression of the attempt on the part of the lenticular cell to reach a stable equilibrium. When the cell reaches a definite size it becomes unstable with respect to its size and divides to overcome this unstability. It renews its youth but is again unstable with respect to the medium. Such a conception of equilibrium is well known to chemists.

Outside of the body the lenticular cells are potentially capable of indefinite growth. In fact, the epithelial cells of the lens may be easily grown in vitro for an indefinite number of generations. In the body the growth and life of the lenticular cells are limited. Many factors regulate this conditional state, such as heredity, food supply, hormones and the growth-retarding influence resulting from surrounding mediums in which the cells are confined. The limitation of cellular growth has been believed to be caused by the exhaustion of some substance essential to growth, by the accumulation of toxic substances and by other internal effects.

Heredity is an important factor in determining the gross growth of the lens. For example, the lens of a mouse will never grow as large as that of a horse. The difference in size between the lens and cataract of Indians and those of Americans and Europeans is probably partly due to heredity. If examples of the gross growth of the lens are taken from vertebrates which have no fixed limit of adult size, as is said to be true for fishes and amphibians, there is seemingly an exception. In this case, the adult lens may apparently grow continuously. However, the factor of size enters into the determination of the growth of the lens of fishes and amphibians; that is, the growth of the whole animal is more influential than the age of the animal in determining the growth of the lens. The same condition probably holds true for the growth of the lens in mammals which have a stunted growth owing to lack of nutrition. However, in normal animals having a determinate growth as soon as a certain size is reached the growth practically stops.

Growth and senescence of the lens occur at the same time. In fact, in certain tissues senescence is regarded as the reciprocal of growth. The lens begins to grow old as soon as it begins to form in the embryo. In the first part of its life changes in growth predominate, and in the last part senile changes predominate. Senescence of the cells is associated with death of the cells. The amount of cytoplasm is greatly increased in proportion to that of the nucleus. The amount of inert metabolic substances, such as albuminoid, increases and the total cellular metabolism is decreased. The cells, weakened by the process of senescence, break down with the unfavorable conditions, and degenerative processes begin.

Heredity is also a factor in determining the senescence of the lens. As the duration of life is influenced by heredity, so is the length of the life of the lens affected. Steinhaus [4] has found that presbyopia, a lenticular sclerosis is related to the life expectancy or biologic age of the body. It is well known that senile cataract tends to occur in persons of a family at the same age. Moreover, senile cataract tends to develop in the two eyes relatively at the same time. Evidently the formation of senile cataracts is best explained by the biologic process of senescence of the lens.

CONCLUSIONS

The process of growth of the mammalian lens follows that of a monomolecular (or autocatalytic) reaction. The normal mammalian lens grows toward a maximum and not continuously in the manner in which it has been assumed to develop. The growth in weight of the lens is dependent on the animal breed (or stock). The formation of a senile cataract is best explained on the basis of a biologic process of senescence.

4. Steinhaus, H.: Untersuchungen über den Zusammenhang von Presbyopie und Lebensdauer unter Berücksichtigung der Todesursachen, Arch. f. Augenh. **105**:731, 1932.

REACTION OF BUFFER SOLUTION AND OF OPHTHALMIC DRUGS

A FURTHER NOTE

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In a previous article by Smith and myself [1] some facts concerning the reaction of solutions used in the eye were pointed out, and the advantages were indicated of using solutions with the following three properties: (1) stability of reaction under ordinary conditions of use, (2) the possible production of solutions of a known reaction within a certain range by simple changes in composition and (3) a bacteriostatic effect by the inclusion of an effective preservative.

It was found that these properties were obtained effectively by the combination of an acid buffer solution with a stock sodium carbonate solution. The addition of 1 cc. of the latter solution to 50 cc. of the former gave a solution with a p_H of 7.6, which we called an alkaline buffer solution.

Since this article was published it has become apparent through our own use of drugs in these solutions and through inquiries of colleagues that further discussion of the subject is indicated.[2]

Since certain drugs, such as phenacaine and butyn, are soluble only in a medium more acid than our acid buffer solution, while fluorescein sodium requires a reaction more alkaline than that of our alkaline buffer solution, it seemed desirable to obtain a greater range of reaction if this could be done simply.

The constituents of our buffer solution included boric acid because of its preservative qualities and potassium chloride as a neutral salt for which certain synergistic properties had been claimed. The strong sodium carbonate solution, in spite of certain theoretical objections, was employed as a stock solution because such small amounts were required to effect the desired changes in reaction without marked changes in volume, which would have complicated matters unnecessarily. It was found that by increasing the amount of boric acid an acid buffer of p_H 5 was

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1. Gifford, S. R., and Smith, R. D.: Effect of Reaction on Ophthalmic Solutions, Arch. Ophth. 9:227 (Feb.) 1933.

2. Correspondence with Miss Edith Heister of Cincinnati, whose views are from the pharmacist's standpoint, has suggested several questions.

obtained, in which phenacaine and butyn were easily soluble. By adding amounts of the stock sodium carbonate solution, reactions of from p_H 5 to p_H 9 could be simply obtained. In making the combinations, 30 cc. of the acid buffer solution was used to approximate the usual 1 ounce prescription, instead of 50 cc. as formerly.

The composition of the new acid buffer solution (no 1) is as follows:

Boric acid (anhydrous)	12.4 Gm.
Potassium chloride (anhydrous)	7.4 Gm.
Distilled water	1,000 Cc.

The composition of the stock sodium carbonate solution is:

Sodium carbonate (anhydrous)	21.2 Gm.
Distilled water	1,000 Cc.

The reactions obtained by adding varying amounts of the stock sodium carbonate solution to 30 cc. of the acid buffer solution are shown in the table.

Reactions on Addition of Stock Carbonate Solution to Acid Buffer Solution

Amount in cc.	0	0.05	0.1	0.25	0.50	1.00	1.50	2.00	3.00	4.00	8.00
Reaction (p_H)	5.0	6.0	6.2	6.75	6.95	7.2	7.6	7.8	8.2	8.4	9.0

It will be seen that this range includes solutions as acid and as alkaline as can be well tolerated by the conjunctiva and cornea, and also those compatible with the solubility of all the common drugs used for instillation into the eye. In deciding which of these combinations will be of advantage for various drugs, the solubilities of these drugs and also the object to be obtained in using buffer solutions as mediums must be remembered. The object is the maintenance of a drug at the reaction least irritating to the eye and most compatible with its local effect and absorption during prolonged daily use.

Thus for phenacaine and butyn, the new acid buffer solution no. 1 (p_H 5) is suitable. This seems to have a definite advantage over our former preparation in which dilute acetic acid was added to our acid buffer solution, since the manufacturers of phenacaine state that the addition of acetic acid produces changes in the alkaloid. Such acid solutions are irritating, but in the case of phenacaine this cannot be avoided, while butyn dissolved in distilled water has a reaction almost as acid (p_H 5.5). The solution in acid buffer is not noticeably more irritating, while the preservation of the drug in this solution is better than in distilled water.

For the zinc salts this solution is also practical, since, according to Lipschütz, they are most effective in acid solution. A less irritating solution is afforded, however, at p_H 6, and this solution, made by adding 0.05 cc. of the stock sodium carbonate solution to 30 cc. of acid buffer no. 1 (or more conveniently, 0.1 to 60 cc.), may be considered

optional for the zinc salts. It may be designated acid buffer solution no. 2. Cocaine and epinephrine are also soluble in this but not in more alkaline solutions.

Most of the alkaloids, as shown in the previous article, are best absorbed, as well as least irritating, in a slightly alkaline solution of p_H 7.6. This, designated alkaline buffer solution no. 1, is made by adding 1.5 cc. of the stock sodium carbonate solution to 30 cc. of acid buffer solution no. 1. It is suitable for atropine, homatropine, pilocarpine and physostigmine. There is some evidence that while marked idiosyncrasies to these drugs are not affected by their use in this medium, certain milder reactions, such as occur with physostigmine and pilocarpine, are less apt to occur when this alkaline solution is employed. For use as a simple eyewash and as a substitute for tears in mild epithelial dystrophy and chronic conjunctivitis in elderly persons, this solution seems ideal, as it is mildly antiseptic and absolutely nonirritating. It has been employed gratefully by many patients for these purposes. The use of a larger amount of boric acid in the new solution here described enhances its preservative powers, which was desirable, since the former alkaline buffer allowed a slight growth of molds after prolonged use.

It must be recognized that the physostigmine salts in this solution turn dark red almost immediately. Some pharmacists, and even the manufacturers, according to Miss Heister, consider that this change of color denotes oxidation and hence loss of potency in the drug. While it must indicate some chemical change, it may be stated that the potency of such red solutions is in no way inferior to that of clear solutions in acid mediums. Such an alkaline solution several months old when tested recently on the pupils of rabbits and of a normal person against a fresh clear solution in saturated boric acid solution produced a more rapid miosis, the difference in the diameters of the two pupils being from 1 to 2 mm. after twenty minutes.

In vernal conjunctivitis, Lehrfeld [3] advised the use of a 2 per cent solution of sodium carbonate to dissolve the stringy secretion almost always present, stating that the effect on the symptoms is marked. It was found that the reaction of such a solution is more alkaline than p_H 9, and that it produces a marked sensation of burning. A patient with keratitis was seen using such a solution, and it seemed that a solution less alkaline than this but more alkaline than alkaline buffer solution no. 1 would be more suitable. Such a solution, of p_H 8.4, is obtained by adding 4 cc. of the stock sodium carbonate solution to 30 cc. of the acid buffer solution. This, called alkaline buffer solution no. 2, has been given for frequent use to a number of patients with vernal

3. Lehrfeld, L.: Sodium Carbonate (Monohydrated) in Treatment of Vernal Conjunctivitis, J. A. M. A. **100**:812 (March 18) 1933.

conjunctivitis, who have used it without complaining of its irritating effect and with definite relief from symptoms. It has also proved useful in other cases of chronic conjunctivitis with tenacious and somewhat dry secretion. A 3 per cent solution of sodium bicarbonate was found to have a reaction the same as this, p_H 8.4, and may be used conveniently by those not wishing to have the buffer solutions prepared.

For the practical use of the buffer solutions by many ophthalmologists, they must be made up by druggists. It was found that when simple directions were given to a reliable druggist the solutions produced corresponded accurately, for practical purposes, with those made in our laboratory. The reactions did not vary more than p_H 0.2 from those of our solutions, and this was not sufficient to affect the solubility of the drugs employed. In the case of phenacaine a slight change to the alkaline side may precipitate the drug, but if the solution is warmed, solution occurs, and the drug has remained dissolved in such solutions for a considerable time. Naturally not every druggist will undertake the trouble of preparing the solutions, so that one will usually choose one druggist capable of doing so for all prescriptions involving their use. He should be given the composition of the two original solutions, which he may keep on hand, and the mixtures of these necessary for the preparation of acid buffer solution no. 2, (0.1 to 60 cc.), alkaline buffer solution no. 1 (1.5 to 30 cc.) and alkaline buffer solution no. 2 (4 to 30 cc.). A fifth solution, of p_H 9 (8 to 30 cc.), is used only to dissolve sodium fluorescein. There is a form of fluorescein soluble in distilled water which, on account of its reaction, is less irritating than the necessarily alkaline solutions of sodium fluorescein. It is called water-soluble fluorescein.

In making up the solutions, it was thought that the variation in reaction of distilled water as ordinarily preserved might affect reactions considerably. It is known that by absorption of carbon dioxide from the air distilled water rapidly loses its neutral reaction and becomes acid. Truog[4] found that it reached a p_H of from 5.6 to 5.8 in ten minutes. As he pointed out, however, the amount of ionizable matter in such solutions is so minute that the reaction is changed by equally minute amounts of any materials added; hence the original acidity of distilled water is of little practical consequence. We found that when our distilled water (p_H 5.8) was boiled to drive off carbon dioxide and cooled in connection with a tube containing soda lime, it was almost neutral (p_H 6.8). Buffer solutions made up with this water showed practically the same reaction as with the ordinary distilled water, the acid buffer being of p_H 5 while the other buffers were identical in reaction with those first prepared. Hence this refinement of technic is unnecessary, the

4. Truog, E.: Science **74**:633 (Dec. 18) 1931.

only precautions necessary being the use of good scales, of clean glassware and of pipets measuring accurately to 0.1 cc. The anhydrous forms of boric acid and the salts must be employed. If it be desired to buffer euphthalmine solutions to lessen their irritating qualities, the manufacturers state that the use of sodium carbonate is undesirable as it may affect the alkaloid. There seems to be no objection to the use of acid buffer solution no. 1 for this purpose. The manufacturers suggested to Miss Heister the following buffer solution as a modification of this:

Boric acid	12.4 Gm.
Sodium borate	0.5 Gm.
Potassium chloride	7.4 Gm.
Distilled water	1,000 Cc.

This, Miss Heister found, had a p_H of 6.5. When made up with euphthalmine to a 5 per cent solution, the p_H was 5.9, considerably less acid than the solution of euphthalmine in distilled water (p_H 4.8), and hence somewhat less irritating.

SUMMARY

1. A new series of buffer solutions is proposed which gives a range of reaction from p_H 4.8 to p_H 9.

2. By the use of four solutions, most of the drugs employed in ophthalmologic practice may be dissolved and satisfactorily preserved during ordinary use.

3. The indications suggested for the four solutions are as follows:

(*a*) Acid buffer solution no. 1: For butyn and phenacaine.

(*b*) Acid buffer solution no. 2: For the zinc salts, cocaine and epinephrine.

(*c*) Alkaline buffer solution no. 1: For homatropine, atropine, physostigmine and pilocarpine; for use as an eyewash and as a substitute for tears in epithelial dystrophy and certain cases of chronic conjunctivitis.

(*d*) Alkaline buffer solution no. 2: For use in vernal conjunctivitis and certain cases of chronic conjunctivitis with tenacious secretion.

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

ACUTE OPTIC NEURITIS IN DEMYELINATING DISEASES OF THE NERVOUS SYSTEM

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In recent years there has been a great deal of interest shown in the so-called demyelinating diseases of the nervous system. In this category are included disseminated (multiple) sclerosis, acute disseminated encephalomyelitis, neuromyelitis optica (Devic's disease) and encephalitis periaxialis diffusa (Schilder's disease). A review of the neurologic literature discloses much that is contradictory and overlapping. The present day identification and classification rest more on clinical differences and slight pathologic variations than on firm etiologic or biologic grounds. It is not impossible that all these conditions are variations of one main entity, and that their clinical differences are due merely to degrees of involvement or to predilection for special tissue. Briefly stated, the current belief is that these diseases are of infectious origin, and that they are probably due to some neurotropic virus which is disseminated by the blood stream or by the cerebrospinal fluid. The common pathologic observations are perivascular infiltration, demyelinization, degeneration of the axis-cylinders and glial activity.

Involvement of the optic nerves, chiasm and tracts may occur in all these diseases. Sometimes there seems to be a distinct affinity for these structures, which is not surprising, considering that they are really brain tracts.

Owing to the anatomy of the optic nerve, it is natural to find the morbid processes in its retrobulbar portion, where the nerve fibers are myelinated. Consequently, it is only when the disease is extensive or when it occurs far forward near the lamina cribrosa that an extension of the acute reaction is visible in the intra-ocular portion of the optic nerve. No one has reported as yet the presence of the disease in the retina, which might happen in the event of a patch of medullated nerve fibers being present. In the demyelinating diseases, when one speaks clinically of acute optic neuritis, one means acute retrobulbar neuritis that has manifested itself by edema and congestion in the papilla. The

ophthalmoscopic picture varies from slight blurring of the margins of the disk to definite swelling of the entire surface. This swelling seldom measures more than 1 or 2 diopters of elevation. Because of the immediate and usually profound loss of vision, the edema of the optic disk seen in these diseases will not be confounded with that of early papilledema due to increased intracranial pressure, in which the visual disturbance comes on later and is gradual.

I propose in this review, first, to discuss demyelinating diseases of the central nervous system, so far as they exhibit what is commonly designated as acute optic neuritis, and, second, to report six cases of such diseases that have come under my observation recently.

DISSEMINATED SCLEROSIS

According to Osler, disseminated sclerosis is one of the commonest organic diseases of the nervous system. As a rule, this disease runs a prolonged downward course and is characterized by remissions. Adie [1] stated that it is the ophthalmologist who first sees nearly one half of the unfortunate persons who suffer from this disease; that in nearly one half of all cases acute retrobulbar neuritis is an early symptom; that in more than 33 per cent it is the first symptom, and that in his series of cases the interval between the attacks of retrobulbar neuritis and the appearance of symptoms elsewhere in the body varied from a few weeks to as long as twenty-four years; the average interval was six and eight-tenths years.

Lillie [2] stated that in five hundred definitely proved cases of disseminated sclerosis observed in his clinic visual disturbances were the primary episodes in 15 per cent, and in from 35 to 40 per cent disturbances of vision occurred as a second or third episode. It seems that females with disseminated sclerosis are more susceptible to retrobulbar neuritis than males with this disease. In the classic descriptions of retrobulbar neuritis in association with multiple sclerosis mention is made of the sudden onset, the central scotoma, the tendency to recovery and the normal fundus picture until temporal pallor of the disks appears. This picture holds true only when the lesion occurs at some distance behind the globe, the so-called neuritis axialis that affects the papillomacular bundle. However, it is in the acute forms, when the lesion is situated just behind the lamina cribrosa, that the visible reaction in the nerve head occurs. It is the general opinion that visible reaction in the nerve is rare in multiple sclerosis, but I believe that with more careful neurologic examinations and with more exact studies of the

1. Adie, W. J.: Tr. Ophth. Soc. U. Kingdom **50**:262, 1930.

2. Lillie, W. J.: Clinical Significance of Retrobulbar and Optic Neuritis. Am. J. Ophth. **17**:110 (Feb.) 1934.

fundus this idea will change. During the last two years my studies of the fundi by stereoscopic photography and by red-free light have led me to believe this.

Uthoff [3] reported five cases of acute optic neuritis observed in one hundred cases of disseminated sclerosis. Buzzard [4] never found any. Brune and Stolting [5] reported two cases of acute optic neuritis seen in a series of thirty-eight cases of disseminated sclerosis. Fleischer [6] found that of thirty cases of acute retrobulbar neuritis without acute reaction in the papilla, twenty-one later showed disseminated sclerosis, and that of twelve cases of acute optic neuritis, five subsequently showed the disease.

As regards other etiologic factors, it is not as difficult to rule out diabetes, excessive use of alcohol or tobacco, metal poisoning and syphilis as it is to exclude the nasal conditions. In the former the development and course are decidedly chronic, the visual disturbances are less and an acute reaction in the papilla is uncommon, whereas in sinusitis the involvement is acute, and papillitis is more common. In late years there has been an increasing tendency to minimize the rôle of infections of the posterior nasal sinuses in the causation of acute optic neuritis.

Campbell [7] reported a case of multiple sclerosis with acute optic neuritis in which vision of the left eye was reduced to perception of movements of the hand. He mentioned the history of previous vague visual disturbances and of pain on pressure over the eyeball during the acute attack. In most of these cases on careful questioning one finds that the acute attack has been preceded by transient visual disturbance. In some instances it is so slight that it almost passes unnoticed. Pain elicited by digital pressure on the eyeball or by rotation of the globe is a common finding during the acute period, and at times deep pain about the eye may be the first symptom of which the patient complains.

Rarely in disseminated sclerosis does acute optic neuritis occur in both eyes simultaneously. Adie [8] stated that bilateral attacks or attacks that come on in the two eyes consecutively are rare, and that their occurrence suggests some other form of inflammation of the optic nerve, such as disseminated encephalomyelitis. The loss of vision in the acute cases of disseminated sclerosis comes with great rapidity. In most instances complete loss of vision occurs within a few hours, and in others there may be gradual diminution extending over a period of a few days.

3. Uthoff: Arch. f. Psychiat. **21**:55, 1890.
4. Buzzard: Brit. M. J. **2**:779, 1893.
5. Brune and Stolting: Monatschr. f. Psychiat. u. Neurol. **7**:184, 1900.
6. Fleischer, B.: Klin. Monatsbl. f. Augenh. **46**:113, 1908.
7. Campbell: Canada Lancet **48**:623, 1915.
8. Adie, W. J.: Brit. M. J. **2**:997 (Dec. 3) 1932.

The first reaction seen in the nerve head is venous engorgement, followed by blurring of the margin of the disk. The blurring of the outline of the disk may be confined to one sector, or may spread until there is haziness of the entire nerve head. In all the cases seen by me the edema began in the upper nasal quadrant. There is hardly ever more than a diopter or two of swelling. Occasionally, one finds a small hemorrhage on or near the disk. The process runs a subacute course for several weeks, gradually subsiding. The result of the acute involvement depends on the amount of destruction of the nerve fibers that has occurred. In some cases it appears as though the nerve head were entirely returned to normal, but careful ophthalmoscopic examination, especially with red-free light and by comparison with the unaffected eye, reveals a slight pallor. When the reaction has been more intense, the color varies from a slight regional pallor to a waxy-white hue of the entire nerve head. An interesting feature in the fundus picture of the resulting atrophy is that the margin of the disk appears sharply defined, as in primary optic nerve atrophy, but there is always a loss of substance. In the most severe cases vision is reduced to the counting of fingers or the perception of light. In some cases, however, the color of the disks does not correspond with the visual loss. Amaurosis is extremely rare.

As the acute attack subsides, in addition to the central and paracentral scotomas, which are almost constantly present, there ensue enlargement of the blindspot and depression of the peripheral field. Later these defects in the field may decrease, but even when vision seems restored to normal, perimetric studies reveal a slight or questionable partial scotoma or a few degrees of contraction of the peripheral part of the field.

Pathologically, the predominant feature of disseminated sclerosis is the perivascular distribution of the "patches." The favorite site is in the tissues immediately surrounding the smaller vessels. The patch constitutes the unit of involvement and runs a course which eventually leads to the formation of the sclerotic plaque. In the early stages there occurs in the perivascular tissue an infiltration of three main types of cells, namely, lymphocytes, plasma cells and the fat granule cells or compound granular cells. The exact origin of the last is not known, but they are thought to be of either lymphocytic or glial derivation (microglia). Their function is phagocytic. Peripheral to the involved perivascular tissue is the area of affected myelin sheaths which undergo dissolution and leave behind degenerated axis-cylinders. The axis-cylinders may remain intact for a long time, and some may even be entirely unharmed, as is evidenced by the return of function. Between the affected area and the normal tissue there is a transitional zone

which limits the process and is composed of a ring of glial cells. As the patch evolves the fat granule cells disappear, leaving dilated perivascular spaces. Fibrogliosis and hyalinization constitute the final changes resulting in the sclerotic plaque. The smaller vessels in the regions affected are generally dilated, but hemorrhages are rarely seen.

The involvement in the optic nerve occurs principally in areas surrounding the vessels. However, extension into the sheaths of the optic nerve has been noted. Most frequently the lesions are found between the papilla and the entrance of the central vessel. In the chiasm and optic tracts the patches are either subpial or subependymal. In discussing the predilection of the virus for the optic nerve, McAlpine [9] expressed the belief that it may pass directly from the ethmoid cells to the optic nerve, or that it may reach the optic nerve from the cerebrospinal fluid in the optic subarachnoid space. However, until more is understood concerning the exciting cause of disseminated sclerosis, it is improbable that the special affinity of the disease for the optic nerve will be explained. Unlike what occurs in syphilis, the nerve fiber layer and the layer of ganglion cells in the retina are usually not affected.

I wish particularly to emphasize that in cases of unilateral acute optic neuritis one must always consider the possibility of disseminated sclerosis, even when no other signs of damage to the nervous system are demonstrable.

ACUTE DISSEMINATED ENCEPHALOMYELITIS

Acute disseminated encephalomyelitis attacks the brain and spinal cord and is characterized by extensive demyelinization. Recent reports indicate that the condition may occur epidemically without any known preceding disease, or that it may follow measles, vaccination or chickenpox. The disease usually begins acutely with symptoms of infection of the upper respiratory tract or of a general infection with headaches, drowsiness, vomiting or convulsions. Within a week or two after the onset, symptoms indicating destructive involvement of the central nervous system develop. Diseases of the third, fifth and seventh nerves are common. Sensory disturbance and muscular weakness appear early. Damage to the cord is evidenced by weakness of the legs, feeble tendon reflexes and sphincteral difficulties. Except for a slight lymphocytic pleocytosis or an increased amount of globulin, the spinal fluid shows nothing distinctive. The mortality rate varies from 30 to 40 per cent. In the patients who survive a definite immunity seems to be established, the condition thus differing from disseminated sclerosis, in which there may be a recurrence. There is also a type of disseminated encephalomyelitis in which the course is not so acute but the lesions are more

9. McAlpine, D.: Brit. M. J. **1**:269 (Feb. 12) 1927.

widespread and death takes place within a few months. There are abortive forms which have a short duration and result in either minimal or no damage.

An outstanding feature in acute disseminated encephalomyelitis is the frequency with which the chiasm, tracts and primary and higher visual centers are affected. Acute involvement of the optic nerve, if present, appears bilaterally, with a marked propensity to reaction in the papilla. Varying degrees of optic nerve atrophy may follow. Studies of the visual fields, particularly during the subacute period of the involvement, may demonstrate, in addition to central scotomas and contracted fields, the presence of hemianopic defects. Recently, Klar [10] described 4 cases of encephalomyelitis optica acuta, none of which proved fatal. He pointed out that there are traits in common with the neuropticomyelitis of the French authors, and that in epidemic abortive cases acute retrobulbar neuritis, generally accompanied by papillitis or choked disk, may be the chief feature. Other disturbances in the nervous system may be slight and unimportant. This fact has caused some authors to make a plea for the separation of disseminated encephalomyelitis from disseminated sclerosis. Knauer and Jaensch, employing the Grüter-Dors technic of corneal inoculation, produced superficial punctate keratitis in rabbits. They used spinal fluid, sputum and brain from patients with encephalitis. In multiple sclerosis the results were negative. Previously, Grüter had produced corneal lesions with herpes virus. Knauer and Jaensch offered this method as a test to facilitate the differentiation of encephalitis from multiple sclerosis. All Klar's cases showed contraction of the field, indicating involvement of the optic tract. Stout and Karnosh,[11] in a study of 28 cases, stated that examination of the eyegrounds usually revealed nothing more than slight hyperemia and haziness of the margins of the disks. In three of their cases definite optic neuritis was seen. In considering the residual symptoms and sequelae of acute disseminated encephalomyelitis, they mentioned a case in which optic neuritis developed eleven months after the acute onset of the disease. They stated that evidence is accumulating to show that the occurrence of a relapse does not exclude the diagnosis of encephalomyelitis. It is the general opinion, however, that relapses do not occur. Morphologically, encephalomyelitis is characterized by the extent and intensity of the demyelinization. Globus [12] pointed out that,

10. Klar, J.: Klin. Monatsbl. f. Augenh. **89**:645, 1932.

11. Stout, R. E., and Karnosh, L. J.: Acute Disseminated Encephalomyelitis: Its Real Clinical Manifestations and Sequelae, J. A. M. A. **101**:667 (Aug. 26) 1933.

12. Globus, J.: Inflammatory Disease of the Central Nervous System: So-Called Nonsuppurative Encephalitis and Encephalomyelitis, Arch. Neurol. & Psychiat. **28**:810 (Oct.) 1932.

despite the widespread distribution of the areas of demyelinization, there are two constant seats: the optic nerve and tracts and the subependymal zone adjacent to the walls of the lateral ventricles.

In the perivascular and adventitial spaces there occur cellular infiltration and glial activity, which are common to all the demyelinating diseases. In this disease there is a wider destruction of axis-cylinders than in multiple sclerosis. In the types that follow vaccination, measles or varicella a distinctive histologic variation is seen. This variation consists in the predilection of the inflammatory reaction for the venules, around which elongated and continuous streaks of infiltration are present; hence the name perivenous encephalomyelitis. Because of this pathologic observation there has been a tendency to separate this form as a special group.

Clinically, in acute encephalomyelitis one may find bilateral involvement of the optic nerve, manifested by optic neuritis and especially by accompanying involvement of the tracts and primary visual centers. In isolated instances recurrent attacks have been reported. The optic nerves may be affected simultaneously, but there have been cases in which an interval as long as two months intervened.

NEUROMYELITIS OPTICA

In 1870, Clifford Allbutt first described a case of acute bilateral optic neuritis associated with acute myelitis. In 1882, Dreschfield reported another. Devic (1894), after whom the disease was named (*maladie de Devic*), called the condition *neuromyélite optique aiguë*. Since then the condition has been described many times, particularly by French authors, under various names, such as *ophthalmoneuro-myélite* and *neuroptico-myélite*. It is interesting to observe how few cases have been noted in the English literature. Holden [13] reported five cases of acute myelitis with retrobulbar inflammation of the optic nerves. In three of these cases varying degrees of reaction in the disks were noted. In two cases there was unilateral involvement of the eyes. He made a point of the fact that in one half of his cases there was lateral hemianopia for one eye alone. Although the fact was not mentioned by him, this would indicate partial involvement of the chiasm or tracts. Beck,[14] in his review of the literature, found that in 25 per cent of the cases the visual disturbances came on before the onset of the myelitis. But Goulden [15] later stated that in 80 per cent of the cases the optic neuritis precedes the appearance of the myelitis. The symptoms and severity of the myelitis naturally depend on the location and amount of involve-

13. Holden, W. A.: Arch. Ophth. **40**:569, 1911; **43**:231, 1914.
14. Beck, G. M.: Brain **50**:687 (Oct.) 1927.
15. Goulden, C.: Tr. Ophth. Soc. London **24**:229, 1929.

ment in the cord. In many instances it seems as though the optic nerves receive the brunt of the attack. In others, particularly those with progressive and ascending myelitis, death may intervene when the process reaches the higher levels in the cord. From 40 to 50 per cent of the persons with this disease succumb. Varying degrees of recovery may occur, and in some cases the improvement is extraordinary.

Uniformly in this condition both eyes are involved, but not always exactly at the same time. The interval between the appearance of the neuritis in the two eyes is seldom longer than from two to three weeks. Often it is a matter of hours. Except for the tendency to bilateralism, the ophthalmoscopic picture differs in no wise from the picture of acute optic neuritis in disseminated sclerosis. Here, also, one must interpret the reaction seen in the disks as being due to the proximity of the focus to the posterior surface of the lamina cribrosa. The sharp outlines of the atrophic nerve heads seen after the acute stage has subsided strengthen this assumption. There is marked loss of vision during the acute stage. The classic axial retrobulbar type seen in disseminated sclerosis, that is, acute optic neuritis without acute reaction in the papilla, is not seen in neuromyelitis optica. Milian, Lhermitte, Schaeffer and Horowitz [16] reported a case with double optic neuritis which ended in death. Postmortem examination of the optic nerve showed heavy and extensive involvement throughout. There were marked perivascular infiltrations and demyelinization. There were numerous streaks in which the nerve fibers were absent. The sheaths were invaded by monocytes. With Bielschowsky's stain the axis-cylinders were seen to be preserved, but they were swollen and in places fragmented. In the chiasm there was glial activity between the fibers. In Beck's case, that of a girl, 15 years old, with bilateral optic neuritis and paraplegia, there were several remissions. Death occurred five years after the onset of the disease. The optic nerves and chiasm showed extensive demyelinization, marked infiltration by lymphocytes and polymorphonuclear leukocytes and cavity formation. On the strength of the clinical picture and these pathologic variations, Beck expressed the belief that neuromyelitis optica, as an entity, differs from disseminated sclerosis. Brain [17] questioned this opinion, stating that this case showed remissions, and that the pathologic variations that were seen were also met with in disseminated sclerosis. He stated that probably neuromyelitis optica and disseminated sclerosis are different forms of the same disease, and that the differences found between them are due to varying degrees of acuteness and intensity. However, until the simi-

16. Milian, Lhermitte, Schaeffer and Horowitz: Rev. neurol. **2**:257, 1931.

17. Brain, W. R.: Critical Review; Disseminated Sclerosis, Quart. J. Med. **23**:343 (April) 1930.

larity or dissimilarity of the agents causing these diseases is found, one is confronted in neuromyelitis optica with a condition in which there is usually bilateral acute optic neuritis associated with myelitis and in which recovery may occur.

ENCEPHALITIS PERIAXIALIS DIFFUSA (SCHILDER'S DISEASE)

In 1912, Schilder [18] described a form of demyelinating disease that occurs chiefly during infancy and childhood, which he named encephalitis periaxialis diffusa. Shelden, Doyle and Kernohan,[19] however, reported a case in a woman of 27 years. There have been definite instances which point to a familial tendency. Pathologically, in the subcortical regions of the cerebral and cerebellar hemispheres there is almost complete destruction of the myelin sheaths, followed by dense replacement gliosis. The occipital and temporal lobes are particularly susceptible. Except in places of intense involvement, the axis-cylinders are spared. A singular point in the histologic picture of this form of demyelinating disease is the absence of mesodermal reaction. Because of this, many think that the disease is degenerative rather than inflammatory. Globus and Strauss [20] would include it in a group of progressive, degenerative, subcortical encephalopathies. Clinically, it is usually characterized by an abrupt onset with gastro-intestinal disturbances. There follow symptoms of general cerebral involvement, such as apathy, blindness (with or without involvement of the optic nerve), deafness, convulsions and spastic paralysis. In young infants the condition may manifest itself by enlargement of the head, which occasionally has been mistaken for hydrocephalus. The disease runs a progressive course, lasting from a few months to two or three years, and ends fatally. I have been able to find in the literature reports of about thirty-five cases. In 60 per cent of these cases, blindness, which may not have been complete in every instance, was noted. In 75 per cent of this number blindness came on as a late symptom. In 90 per cent of the cases with amaurosis bilateral optic nerve atrophy was noticed before death. Some reports mentioned the fact that there was early impairment of vision followed by optic nerve atrophy some weeks later. But there was no ophthalmoscopic report as to the condition of the fundi during the period of "impairment of vision." In only two instances was acute optic neuritis definitely mentioned, once in the report of Schilder's cases and again in the report

18. Schilder, P.: Ztschr. f. d. ges. Neurol. u. Psychiat. **10**:1, 1912.

19. Shelden, W. D.; Doyle, J. B., and Kernohan, J. W.: Encephalitis Periaxialis Diffusa, Arch. Neurol. & Psychiat. **21**:1270 (June) 1929.

20. Globus, J. H., and Strauss, I.: Progressive Degenerative Subcortical Encephalopathy (Schilder's Disease), Arch. Neurol. & Psychiat. **20**:1190 (Dec.) 1928.

of Shelden's second case. Postmortem examination in Shelden's case showed not a single normal myelin sheath in the optic nerves. Histologic studies made in some of the cases of Schilder's disease showed a severe reaction and destruction of the oligodendroglia.

CONCLUSIONS

In demyelinating diseases of the central nervous system, an acute reaction may occur in the optic papilla (acute optic neuritis). Although, in the present stage of knowledge of the demyelinating diseases there seems to be little histologic difference among them, clinically certain well defined types appear. In acute disseminated or multiple sclerosis there seems to be a tendency for a unilateral appearance of acute optic neuritis, with a decided tendency to remissions. In neuromyelitis optica bilateral involvement of the optic nerves, associated with myelitis and with a decided tendency to recovery, is the rule. Acute encephalomyelitis likewise may be characterized by bilateral involvement of the optic nerve, associated with a special disposition to involvement of the optic tracts and with little or no tendency to recurrences. It appears that in Schilder's disease, which is generally fatal in youth, acute optic neuritis is not seen so frequently as in other types of demyelinating diseases. Here the process principally affects the higher centers. In acute encephalomyelitis and neuromyelitis optica the tendency is toward extensive involvement of the optic nerve, and consequently acute optic neuritis appears frequently. In disseminated multiple sclerosis, in which the lesions are more circumscribed, the classic type of axial retrobulbar neuritis occurs, that is, neuritis without reaction in the nerve head, save in the types in which the patch is situated just behind the lamina cribrosa, in which case a visible reaction (optic neuritis) may be observed ophthalmoscopically. This reaction may be slight and is often not diagnosed. I believe that acute optic neuritis occurs more frequently in disseminated sclerosis than a study of the literature indicates. Appearing as the primary episode in the disease, it has repeatedly been ascribed to other conditions, chiefly to infections of the posterior nasal sinuses.

Finally, special attention is called to the susceptibility to involvement of the visual pathways in all the so-called demyelinating diseases of the nervous system and to the fact that in certain instances these structures may be the only ones giving clinical evidence of the presence of these diseases.

REPORT OF CASES

CASE 1.—*Acute optic neuritis in disseminated sclerosis.*

History.—R. H., a man, came to the general clinic of the New York Hospital in May 1932, complaining of numbness and tingling of the right hand, which gradually progressed until the entire right side of the body became numb. No motor weakness was noted, although handwriting became difficult. The entire

episode lasted about one month, and the condition disappeared spontaneously. The abdominal reflexes were diminished, and slight astereognosis was noted. The eyes were normal. A diagnosis of neurosis was made, although it was believed that multiple sclerosis could not be definitely excluded.

On Jan. 30, 1933, the patient was again seen in the ophthalmic department of the New York Hospital. He stated that during the preceding week he had noticed progressive and marked blurring of vision in the left eye. Five days before he had noted a definite sensation of pain in the left eye, not only to touch, but also when rotating the eye. This was followed by blurring of vision, which gradually decreased to bare perception of movements of the hands.

Physical Examination.—This revealed a well developed and well nourished white man, evidently restless and nervous in disposition. His temperature was 36.8 C. (101.8 F.); the pulse rate was 82, the respiratory rate, 20, and the blood pressure, 124 systolic and 66 diastolic. There was no sinusal tenderness.

Ophthalmic Examination.—The right eye was essentially normal. In the left eye there was a slight injection of the temporal portion of the sclera. The pupils reacted normally, and there was no nystagmus. The mobility of the eyeball was slightly restricted, and there was marked tenderness on pressure over the left globe, with pain on voluntary movement. Vision was reduced to perception of movements of the hands. The media were clear. Examination of the fundus showed that the margins of the disk were almost completely obliterated by a surrounding haziness of about 2 diopters of elevation. The edema was less marked on the temporal side. The vessels were tortuous and the veins engorged. There was a small linear hemorrhage in the retina, situated at the nasal margin of the disk.

The pharynx was moderately injected, exhibiting several patches of lymphoid tissue. There were numerous slightly enlarged cervical nodes. The heart, lungs and abdomen were normal. There was no impairment of the cranial nerves or of the reflexes, sensation or coordination.

Laboratory Examination.—The urine was normal. A blood count showed: red cells, 5,700,000, with 16.2 Gm. (112 per cent) of hemoglobin, and white cells, 12,000, with 73 per cent polymorphonuclears. A smear was normal. The blood serum was normal. The spinal fluid pressure was normal; the fluid was clear, with 35 lymphocytes and no globulin. The Wassermann and Kline tests gave negative results. The colloidal gold curve was 1233210000. A pharyngeal smear and culture were negative for diphtheroid organisms and positive for hemolytic streptococci. The sedimentation rate was 38. Examination of the visual fields showed almost complete loss of the left field. The right field was normal. Roentgenograms of the skull, teeth and sinuses showed no abnormality.

Diagnosis.—This was multiple sclerosis with neuritis of the left eye.

Course.—On the fourth day the patient complained of sore throat with dysphagia. His temperature rose to 38.2 C. (100.7 F.); leukocytes numbered 18,000, with 68 per cent polymorphonuclears. The pharynx and tonsils appeared inflamed, with areas of follicular white patches. Under treatment with gargle and painting with 10 per cent mild silver protein and 10 per cent silver nitrate this condition subsided within four days.

On the twelfth day after admission the patient felt definitely improved and noted some return of vision in the left eye. By the time of discharge he was able to perceive the outline of the examining physician readily. Examination of the left fundus showed definite diminution in the haziness of the optic disk, and the hemorrhage was absorbed. The pharynx appeared only slightly injected.

The patient was last seen on March 21, 1933. Vision at this time in the right eye was 20/20—; with + 0.50 — 1.00 cyl., ax. 120 it was 20/15—, and the patient could read Jaeger's test type no. 1 at 13 inches (33.02 cm.). Vision in the left eye was 20/40—; with +0.50 cyl., ax. 90 it was 20/30, and the patient could read Jaeger's test type no. 1 at 13 inches.

The media were clear. The right fundus was normal; the left was pale, with a slight blurring of the upper nasal margin. The left blindspot was slightly enlarged, and there was a relative central scotoma demonstrable with a 0.5 mm. colored test object on the Lloyd stereocampimeter. There were no definite neurologic findings at this time.

CASE 2.—*Acute optic neuritis in disseminated sclerosis.*

History.—A. E., a woman, aged 45, came to the clinic of the New York Eye and Ear Infirmary on May 4, 1933, complaining of sudden loss of vision in the left eye. The patient stated that during the preceding four years she had had attacks of numbness and unsteadiness of certain portions of the body, occurring about once a year and lasting for a few weeks. There was complete recovery between attacks. She had had a similar attack, particularly with weakness of the left side, two weeks previously. There had not been visual disturbances with any previous attack.

Ophthalmic Examination.—In the right eye vision was 20/50 and with a + 1.00 sph. 20/30+. The media were clear, and the fundus was normal. In the left eye vision was reduced to perception of fingers at 1 foot (30.48 cm.). The media were clear. The margin of the disk was very hazy and swollen to about 1 diopter, and the veins were engorged. No hemorrhages were seen. A slight lateral nystagmus was elicited. The blood pressure, urine and spinal fluid were normal, and a roentgenogram of the skull showed a normal picture. The Wassermann reaction of the blood was negative. The patient was referred to a neurologist, who stated: "Because of the optic neuritis, nystagmus, ataxia and the extremely characteristic remissions, I think that the diagnosis of multiple sclerosis is fairly assured."

Course.—On May 18 the patient felt much better. The weakness of the left side had almost completely disappeared. Vision in the left eye was now 20/70 unimproved. The edema of the nerve head was less, and the margin of the disk could be made out. The fields showed a partial central scotoma, with about 20 degrees of concentric contraction of the peripheral parts.

On June 5, the patient was seen again. Vision had improved to 20/50— corrected. She was able to read Jaeger's test type no. 1 at 10 inches (25.4 cm.). The fields were unchanged. However, definite pallor of the entire left disk was noted. After this visit the patient did not return to the clinic, and efforts to establish contact through the social service department were unsuccessful.

CASE 3.—*Bilateral acute optic neuritis in encephalomyelitis.*

History.—A. B., a white man, aged 40, a laborer, reported to the ophthalmic clinic of the New York Hospital on Jan. 2, 1934, stating that at the time of a severe cold three weeks before, he had begun to have dull aching "bone" pains in the lower extremities, accompanied by numbness and indefinite sensory changes. Three days later the eyeballs began to ache, especially on movement. This was followed by rapid blurring of vision in the left eye. A few days later there was some blurring of the right eye.

Ophthalmic Examination.—The extra-ocular movements were normal. Deep pressure over the globes elicited pain. There was no nystagmus. The pupils were

equal and reacted to light and in accommodation. Vision of the right eye was 10/200 unimproved; that of the left was 20/40 unimproved.

The right fundus revealed well developed optic neuritis. The margin of the disk was obliterated, and the veins were engorged. There was about 1 diopter of swelling of the disk, but no hemorrhages or exudates were seen. The left fundus showed definite blurring of the margin of the disk nasally, with venous engorgement. In the right eye there was absolute central scotoma, but with a large test object there was no apparent contraction of the peripheral part of the field.

Diagnosis.—This was bilateral acute optic neuritis.

Course.—Because of acute coryza, the patient was referred to the department for diseases of the nose and throat, where a diagnosis of acute ethmoiditis was made. Bilateral ethmoidectomy was performed. A week later the patient complained that he had lost the ability to increase intra-abdominal pressure when he wished to urinate or defecate. The ophthalmic condition remained unchanged. He was then referred to the neurologic department and was admitted to the hospital on February 16.

Examination.—The temperature was 37 C. (98.6 F.), the pulse rate, 86, and the respiratory rate, 20. The blood pressure was 120 systolic and 70 diastolic. There were complete anosmia, bilateral optic neuritis and weakness on the right side of the face, including the forehead. The tongue deviated slightly to the right and the uvula to the left. The gag reflex was lost. The tendon jerks were less active in the left arm than in the right. Below the second and third dorsal segments there was hypesthesia to pinprick and light touch. The abdominal and cremasteric reflexes were absent. No evidence of motor weakness was found. There was urinary and fecal incontinence.

Laboratory Examination.—The red blood cells numbered 3,070,000, with 75 per cent hemoglobin. The white blood cells numbered 10,530, with 35 per cent immature polymorphonuclears. The blood serum, urine and stools were normal. The spinal fluid was essentially normal, except for the presence of 28 cells; there were 92 per cent lymphocytes and 8 per cent polymorphonuclears.

Summary.—There were disseminated lesions of the optic nerves, the left hemispheres and the white matter of the cord below the third and fourth thoracic segments.

Diagnosis.—This was acute disseminated encephalomyelitis.

Further Course.—On February 21, the patient's condition was unchanged. He was still incontinent. On February 23, his temperature was normal, and his condition was improving. Vision in the right eye was 20/200 unimproved, and that in the left eye was 20/30. The edema of the right optic disk was considerably less, and the left disk was almost normal, except for a suggestion of pallor. The condition of the patient continued to improve, and he was last seen on June 28, at which time the edema of the right optic disk was practically gone, leaving a pale nerve head. Vision in this eye was 20/100 unimproved. There was a central scotoma, but no definite contraction of the peripheral part of the field. Vision in the left eye was 20/30+, with a faint suggestion of temporal pallor. There was a questionable central scotoma for red. The peripheral part of the field was normal. There was no nystagmus, and extra-ocular movements were normal. The facial condition was unchanged. The patient's sphincteric control was greatly improved, and he was no longer troubled by frequency or bed-wetting.

CASE 4.—*Bilateral acute optic neuritis in disseminated encephalomyelitis following varicella.*

History.—W. P., a boy 6 years old, two weeks after an attack of chickenpox suddenly showed weakness of the face and slight ataxia. There was a tendency to fall to the right side as though the right foot were weak. This was followed by bilateral convergent squint. Two weeks later, on March 30, 1933, he complained of blurred vision and began to vomit after meals. He was admitted to the New York Hospital on April 1.

Examination on Admission.—The temperature ranged from 99 to 100 F.

Ophthalmic Examination.—There was bilateral palsy of the abducens nerve. The left pupil was slightly larger than the right. Both reacted to light and in accommodation. The left optic disk was swollen about 1 diopter, and the veins were markedly engorged. The outline of the right disk was slightly blurred nasally, and the veins were slightly engorged. Lateral nystagmus was noted.

Examination further revealed facial palsy on the right and ataxia of the body and legs. There were general hypotonicity and bilateral adiadokokinesis. The deep reflexes were hyperactive. The Romberg sign was positive. Examination of the spinal fluid showed a pressure of 150, 1 cell and 110 mg. of sugar per hundred cubic centimeters. A roentgenogram of the skull showed no abnormality.

Diagnosis.—This was acute encephalomyelitis following varicella.

Course.—On April 10, the condition remained unchanged. The parents decided to take the child home. On April 21, because his condition was worse, the child was brought back to the hospital. At this time he was unable to walk, and the vomiting continued. The right eye was inflamed. The patient held his head flexed to the chest.

Ophthalmic Examination.—In the right eye there were considerable conjunctival injection and marked ptosis; the cornea showed keratitis neuroparalytica. The fundus was not seen satisfactorily. In the left eye the edema of the disk was less, and the margin was distinct, but the entire disk was pale. There was bilateral sixth nerve palsy.

Neurologic Examination.—The cooperation was poor. No evident sensory changes were present. There was no evident difference in the motor responses of the two sides. Bilateral sustained ankle clonus was present. Examination also showed bilateral internal strabismus, ptosis of the right eyelid, facial palsy on the right, absence of the corneal reflexes, definite ataxia, general hyperactivity of all the reflexes, a chronic Babinski sign, a positive Oppenheim sign and no Kernig or Brudzinski sign. The intracranial pressure was not increased. The diagnosis of acute encephalomyelitis following varicella was confirmed.

On April 30, the condition of the patient became worse. He was incontinent and sweating, and his speech was less intelligible. The abdominal and cremasteric reflexes were absent. The Babinski reflex was positive.

Lumbar Puncture: The spinal fluid pressure was 140 mm. of water. There was no block. The fluid was clear, and there was no globulin. Sixty milligrams of sugar per hundred cubic centimeters was present.

Further Course.—On May 14, hypostatic pneumonia developed, and the patient was apathetic. The respirations became slower, and he expired at 3 p. m. Permission for postmortem examination was refused.

CASE 5.—*Bilateral acute optic neuritis in neuromyelitis optica.*

History.—I am mentioning this interesting case through the courtesy of Dr. E. D. Friedman, who will report it in greater detail later.

E. W., a girl 9 years old, had pain in the back accompanied by vomiting on Oct. 9, 1932. She was operated on for acute appendicitis under general anesthesia. The day following the operation she complained of a feeling of numbness in the legs. Two days later she was unable to move her arms. Examination at this time showed the eyes to be entirely normal, with no nystagmus. There was complete paralysis of the lower extremities, with absence of the abdominal and Babinski reflexes. Below the level of the fourth dorsal segment there was disturbance of all the sensory reflexes. The temperature was normal. The patient was incontinent. Lumbar puncture showed a clear fluid and 26 cells. A roentgenogram of the spine showed nothing abnormal.

Diagnosis.—This was transverse myelitis at the level of the fourth dorsal segment.

Course.—From this time on there was steady improvement, with return of most of the muscular powers. On December 30, two months later, the patient complained of blurring of vision, particularly in the left eye, extending over a week. She was able to walk into the office unassisted. The left pupil was enlarged and fixed. The right reacted sluggishly to light. The left eye was amaurotic. Vision in the right eye was unaffected. Ophthalmoscopic examination of the left fundus showed typical optic neuritis. The disk was swollen and slightly raised, with blurred outlines and marked venous engorgement. The right fundus was normal.

The patient was admitted to the Beth Israel Hospital on Jan. 3, 1934. A few days later vision in the right eye became blurred. Examination showed neuritis in the right eye similar to that in the left. There was a return of the spinal lesion at the level of the fourth dorsal segment, with paraplegia and sphincteric difficulties.

Within a few weeks the edema of the left optic disk receded, leaving the disk pale and definitely atrophic. Vision improved in this eye, and the patient could now see movements of the hands. With a large test object there were a definite central scotoma and some suggestion of contraction of the peripheral portion of the field. This was followed by improvement of vision in the right eye. The edema of the disk in this eye began to recede. On February 15, the patient was able to count fingers with each eye, read the largest test type and recognize colors. Both disks were whitish, and the margins were still somewhat blurred.

At this time, however, another focus at the level of the fifth cervical segment and also a Horner syndrome on the right side developed. On March 4, the condition was unchanged. There was now bilateral optic nerve atrophy, and the nerve heads showed a loss of substance, but the margins of the disks were distinct. The vessels were not markedly attenuated. Vision in both eyes was reduced to perception of movements of the hands. The pupils were large and fixed.

From this time on the patient's general condition became worse, and she expired on June 18. Permission for postmortem examination was refused.

CASE 6.—*Bilateral acute optic neuritis in encephalitis following measles.*

History.—On March 19, 1933, C. S., a girl, 6 years old, contracted measles in a mild form. There were a characteristic rash, conjunctivitis, Koplick's spots and fever. All the symptoms disappeared within seven days, and she was about to be allowed up when, on March 27, she complained of pains over the right eye, followed by sudden loss of vision. Within six hours the left eye presented similar

signs, and the child was just able to perceive light. On the day previous to the onset of the visual disturbances she had vomited projectilely three times. The temperature was normal. The condition was diagnosed as encephalitis following measles.

The child was referred to the New York Nursery and Child's Hospital on March 30, where I saw her in consultation with Dr. Harold T. Vogel.

Ophthalmic Examination.—This showed questionable perception of light in each eye. The pupils were 3 mm. in width and did not react to light. The media were clear. There was a slight blurring of the margin of the disk in each eye, especially nasally and below, and the veins were full. A diagnosis of bilateral optic neuritis was made.

Neurologic Note (Dr. T. K. Davis).—There was a question of paresis of the upward gaze. The pupils were dilated and inactive. There was a slight weakness of the right arm and leg, and the abdominal reflexes were less on that side. The lesion was not in the occipital poles. Instead, one could be fairly sure of its localization in the region of the corpora quadrigemina, involving the right peduncle. The impression was that of measles encephalitis.

Course.—During the following week both optic disks became entirely obscured by edema. There was an elevation of about 1 diopter in each eye. The veins were markedly engorged, but no hemorrhages were noted. Two weeks after admission to the hospital, on April 14, vision began to improve. The patient could now count fingers at 6 inches (15.24 cm.) with each eye. The edema of the disks had receded, and the veins were less engorged. By indirect illumination beginning pallor of the nerve head was noted.

Further Neurologic Note.—On April 17, the child had improved markedly. The upward gaze had returned; the pupils were enlarged (atropine); the edema of the optic disks was receding; hearing appeared normal on each side, and the neck was not stiff. The deep reflexes were all present, and the superficial reflexes were active and alike on both sides. There was a slight choreiform unsteadiness in the hands when outstretched, a condition which was noted occasionally also when the child was not relaxed. The findings accorded with a diagnosis of convalescent encephalitis (measles).

Further Course.—The patient was discharged from the hospital a week later. Examination showed vision in the right eye to be 8/200 unimproved and that in the left eye to be 10/200 unimproved. The patient could read the largest Jaeger test type with difficulty. The fields showed definite central scotomas, especially for colors, with about 10 degrees of concentric contraction of the peripheral portions. The pupils were both 2.5 mm. in diameter and reacted to light. The fundi revealed bilateral optic nerve atrophy. The disks were very pale, and the margins were well delineated; there was loss of substance, but the vessels were not noticeably constricted.

Correspondence

PELLAGRA AS A CAUSE OF OPTIC NEURITIS

To the Editor.—Relative to Dr. Joseph Levine's article on pellagra as a cause of optic neuritis, which appeared in the December issue of the ARCHIVES (12: 902, 1934), may I answer Dr. Levine's query concerning the changes in the fundi in cases of pellagra by stating that I do not know of any published work on the subject? It happens that in my work as consultant to the South Carolina State Hospital for the Insane I see a fair number of cases of pellagra. An examination of the eyegrounds is required for every patient admitted to the hospital. My impression is that there are no typical changes in the eyegrounds of pellagrins.

A great many pellagrins have other pathologic conditions along with pellagra. There have been several cases in which there were degenerative retinal and choroidal conditions which were thought to be due to a toxic focus of infection rather than to any dietary or vitamin deficiency, or whatever is supposed to be the cause of pellagra.

About twenty years ago there was an international conference on pellagra in Columbia, S. C. The late Dr. J. W. Babcock, who collaborated with Dr. Lavinder of the United States Public Health Service in a book on pellagra, was at that time head of the state hospital and much interested in all phases of the subject. The ophthalmologist at the hospital, who at that time was Dr. E. M. Whaley, studied the fundus oculi of 240 pellagrins. He reported a variety of conditions but nothing that was typical or pathognomonic of pellagra.

I have inquired of three or four ophthalmologists in Columbia, and they all seem to be of the same opinion, namely, that there is no typical picture in the fundus in cases of pellagra. The work of Dr. Whaley was not published, but it was reported at the convention. This information was obtained from Dr. T. M. DuBose Jr., a physician in Columbia, who assisted Dr. Whaley in examining this series of patients.

Dr. Simon R. Lucas, of Florence, S. C., stated that he has never found any particular outstanding lesion in the fundi of pellagrins but that he expects to make a more careful examination of these patients in the future.

WALTER J. BRISTOW, M.D., Columbia, S. C.

News and Notes

SOCIETY NEWS

Atlantic City Session of American Medical Association.—The Section on Ophthalmology of the American Medical Association has appointed the following section exhibit committee for the scientific exhibit at the Atlantic City session of the American Medical Association and the Canadian Medical Association: Georgiana Dvorak-Theobald, Oak Park, Ill., chairman; Wilfred E. Fry, Philadelphia; Parker Heath, Detroit, and Alexander E. MacDonald, Toronto, Canada. Application for space must be made by Feb. 25, 1935. Application blanks may be obtained from any of the members of the committee or from the Director, Scientific Exhibit, 535 North Dearborn Street, Chicago.

Mr. Leslie Paton of London will be the guest speaker at the next meeting of the Section on Ophthalmology of the American Medical Association in Atlantic City, June 1935.

British Medical Association.—The annual meeting of the British Medical Association will be held at Melbourne, Sept. 11 to 13, 1935. The president is A. J. Ballantyne, Glasgow. The vice-presidents are: J. Lockhart Gibson, Brisbane; R. Foster Moore, London; F. G. Antill Pockley, Sydney, and H. M. Traquair, Edinburgh. The honorary secretaries are: J. Ringland Anderson, Melbourne; Max Yuille, Melbourne, and J. H. Doggart, London.

Ophthalmologic Society of Belgium.—A meeting of the ophthalmologic Society of Belgium was held on November 25 at Brussels.

Ophthalmologic Congress in India.—The 1935 meeting of the All India Ophthalmological Society will be held at Madras, under the presidency of Colonel Duggan.

UNIVERSITY NEWS

Dr. S. Ernest Whitnall, professor of anatomy at McGill University, has resigned to accept a similar professorship at the University of Bristol, England.

Obituaries

HOWARD FORDE HANSELL, M.D.
1855–1934

Dr. Howard Forde Hansell, an ophthalmologist of international reputation, died in Philadelphia on Nov. 5, 1934.

He was born in Philadelphia on Oct. 25, 1855, the son of Barnet Hansell and Rebecca Wetherill Hansell, both of English lineage. His grandfather, William S. Hansell, founded the saddlery business on Market Street in Philadelphia, while his grandfather on his mother's side was a prominent drug merchant on North Front Street, which was at that time a leading business center.

Dr. Hansell obtained his early education in the public schools of Philadelphia, graduating from the Central High School in 1873. He entered Brown University in the fall of that year and pursued his studies with diligence, receiving the degree of A.B. in 1877 and that of A.M. in 1880. As an undergraduate at Brown University he associated with many young men who were destined to occupy places of prominence in all walks of life—T. Aldrich, cotton manufacturer and benefactor of Brown University; Charles Howard Johnson, late Chief Justice of the Rhode Island Supreme Court; William Paine Schiffield, former Congressman from Rhode Island; Rev. Willis Frye Thomas, D.D., noted British missionary to Burma; Judge William Grant Van Horne of the International Court of First Instance, Cairo, Egypt, and Fred Homer Williams, late trustee of Brown University and prominent Boston lawyer. His roommate was the late Rathbone Gardner, at one time leader of the Rhode Island bar. He was a member of the Psi Upsilon fraternity.

The final year at Brown University was devoted mainly to biology as a preparatory step to the study of medicine. He matriculated at Jefferson Medical College in the fall of 1877 and became a private student of Dr. J. C. Wilson, recently deceased. In 1879, he received his degree. Following graduation he was appointed assistant in the medical clinic under Prof. J. M. Da Costa and also in the ophthalmologic clinic under Prof. William Thompson.

In 1880, Dr. Hansell went to Europe to pursue further his studies in ophthalmology, to which he had decided to devote his entire attention. In Berlin he received clinical instruction under Hirschberg, and in Vienna, under Fuchs, Stellwag and Arlt.

On his return to Philadelphia he was appointed clinical assistant under Professor Thompson in the department of ophthalmology and was later promoted to the position of chief of the clinic.

Coincidentally with his advancement at Jefferson Medical College, Dr. Hansell joined the staff of the Polyclinic Hospital. He later became professor of ophthalmology at that institution and was elected president of the faculty for four successive years. He also received the appointment of attending ophthalmologist at the Philadelphia General Hospital, a position which he held for more than twenty years.

In 1904, he received from the board of trustees of Jefferson Medical College the appointment of professor of ophthalmology, succeeding Dr. George de Schweinitz. In 1926, following his resignation, he was made emeritus professor of ophthalmology.

Dr. Hansell contributed generously to various ophthalmic journals throughout his career. Among some of his more important papers may be mentioned: "History and Study of the Practice of Refraction of the Eye in Philadelphia"; "Some Observations in Ametropia and Heterophoria"; "Luetic Ophthalmitis"; "Treatment of Retinal Detachment;" "Accommodation in Myopia"; "Healthy Habits in Reference to Preservation of Vision"; "High Hyperopia from a Clinical Standpoint"; "Report of Fifty Cases of Homer-Smith Preliminary Cystotomy," and "A Brief Report of Four Cases of Atypical Retinitis Pigmentosa." The last paper was read before the Oxford Congress in 1924 and received wide and favorable comment.

His major literary works were: "A Manual of Clinical Ophthalmology," in collaboration with Bell, published in 1892; "A Practical Handbook on the Muscular Anomalies of the Eye," in collaboration with Reber, published in 1899, and "Text-book of Diseases of the Eye," in collaboration with Sweet, published in 1903.

In 1920, he was a member of the committee (with Howe and Schneidemann) appointed by the Section on Ophthalmology of the American Medical Association to report on the selective investigation concerning the ocular muscles.

He was also a member of the Pennsylvania State Medical Society and the Philadelphia County Medical Society, the American Medical Association and the American Ophthalmological Society. He was elected a fellow of the College of Physicians of Philadelphia in 1886 and served as chairman of the Section of Ophthalmology from 1907 to 1908, inclusive.

His private practice became extensive in the course of time, and yet each patient, regardless of his station in life, received every consideration. He was scrupulously honest with his patients, as he was in all other matters. Any evidence of quackery that came to his attention received his utter condemnation.

Dr. Hansell was quiet, reserved and studious, yet he possessed a keen sense of humor.

His lectures, which were always carefully prepared, were well attended by the students. I can recall his interesting presentation of

the cases to the class during my senior year, interspersed with witticisms that brought considerable amusement to the members of the class. He always spoke clearly and with ease. Rarely has a lecturer been more highly respected by the student body.

Dr. Hansell married Emilie Read Vogdes, the daughter of General Israel Vogdes of the United States Army, on Jan. 3, 1887, in Grace Church, New York. This marriage proved an exceedingly happy one. One son, Howard Forde Jr., was born of this union.

For many years it was the custom of Dr. Hansell to travel abroad during the summer months, visiting the various European clinics. In 1914, he visited the clinic of Stanculeanu in Bucharest, Roumania, to make a special study of his method of extraction of cataracts. On the way home he and Mrs. Hansell were temporarily held up in Russia by the outbreak of the World War, and his recital of the trials and tribulations that beset them during their escape and return to the United States unfolded a stirring narrative.

With his retirement from active practice he decided to spend the greater part of his remaining days in the cities abroad to which he had become attached during his earlier travels. He had only recently returned from Europe when he was stricken with his final illness.

The death of Dr. Hansell came as a distinct shock to a host of medical friends and admirers, not only in Philadelphia but throughout the country, for, though of a decidedly retiring disposition, he had by years of earnest, kindly living endeared himself to many both in and outside of the medical profession.

CHARLES E. G. SHANNON, M.D.

JOHN DUNN, M.D.

1862–1934

On Nov. 7, 1934, Dr. John Dunn, aged nearly 72, died at his home in Richmond, Va. In his passing away, ophthalmology and humanity suffered a grievous loss.

Dr. Dunn was born on Nov. 14, 1862, in Petersburg, Va., where both of his parents were widely known and much loved.

After attending the well known preparatory school of Capt. Gordon McCabe, in Petersburg, he entered the University of Virginia, at Charlottesville. In 1884 he obtained the M.A. degree, and two years later, his M.D. After serving as an intern in the Municipal Charity Hospital of New York City for a year, Dr. Dunn completed a course of special study in Berlin, Paris and London. He was thus unusually well equipped for his special work in medicine.

After his return from abroad Dr. Dunn established his residence in Richmond, where his fine personal character and his professional talents were soon recognized.

In 1893 the University College of Medicine was founded in Richmond. Dr. Dunn became a member of its original faculty, and he also served as treasurer of the corporation, displaying great prudence and foresight. In 1913 the University College of Medicine was merged with the Medical College of Virginia. Dr. Dunn continued to be a member of the new faculty until 1923, when he was forced to resign because of impaired health. Altogether, he had taught in the two schools for a period of thirty years.

As senior warden of the All Saints Episcopal Church, Dr. Dunn was much interested in the activities of the church. He was also a member of several scientific societies, and he contributed articles to the leading medical journals. In the earlier issues of the ARCHIVES OF OPHTHALMOLOGY, his name appeared frequently as a contributor.

On June 6, 1894, he married Miss Nellie Porterfield of Nashville, Tenn. She, with Dr. Dunn's four brothers and one sister, survive him. Over the congenial and happy life of Dr. and Mrs. Dunn fell a dark shadow. Their son and only child lost his life in a training camp of the Royal Canadian Flying Corps shortly before the United States entered the World War. Dr. Dunn bore this catastrophe with the fine courage so characteristic of him, and he devoted himself still more to his unselfish efforts to help others.

Dr. Dunn was a tireless student, and it is said by his colleagues that few neurologists equaled him in his profound knowledge of the anatomy and function of the central nervous system. Aside from

medicine, his mind was enriched with the best in literature. A keen observer and a lover of nature, he was an authority on the native orchids of the United States. He was also very fond of shooting. Dr. Dunn possessed a rare sense of humor, and his mind was a fine and well controlled instrument.

Six years before his death he suffered a slight stroke, but he continued to practice for three more years. Then his health failed, and he was forced to give up his professional work. He was made of such fine stuff that though broken in heart and in body he carried on cheerfully, valiantly and unselfishly until the last. A talented, intellectual and kindly gentleman, he was much admired and loved by his colleagues and by his other friends.

"To live in hearts we leave behind is not to die."

WILLIAM H. WILMER, M.D.

Abstracts from Current Literature

Anatomy and Embryology

ANATOMIC LINEAR CLEAVAGE DEMARCATION OF THE SCLERA. W. KOKOTT, Klin. Monatsbl. f. Augenh. **92:** 177 (Feb.) 1934.

The function and the structure of the sclera are of importance in the pathogenesis of glaucoma and myopia. Research has been done on histologic, physicochemical, elastometric and morphologic bases.

The human eye presents a cleavage different from that of the animal eye owing to a difference in the insertion of the extrinsic muscles. The anterior portion of the sclera up to the equator is formed by circular cleavage and fibers, and the posterior calotte by meridional fibers, which bear reference to the insertion of the four recti and the two oblique muscles. Four meridional waves of cleavage and fibers running toward the posterior pole of the globe seem to correlate with the circular system around the optic nerve. It is impossible to give an explanation of the functional significance of the distribution of the fibers of the sclera.

K. L. STOLL.

Aqueous Humor

KRUKENBERG'S SPINDLE. F. CARAMAZZA, Arch. di ottal. **41:** 1 (Jan.-Feb.) 1934.

Caramazza gives a complete review of the literature, including sixty-four reported cases. He reports two additional ones. Both occurred in women over 50 years of age. One patient was myopic (1 diopter) and had normal vision. The slit lamp showed the typical granules of pigment on the corneal endothelium arranged in a spindle 3.5 mm. long and 2 mm. wide in the middle. Other granules of pigment were seen at some distance from the spindle on the cornea and lens capsule. The iris showed no signs of depigmentation. The second patient showed moderate myopic astigmatism. The spindles resembled those in the first case. The iris showed slight depigmentation of the pupillary border and the vitreous was fluid.

The author believes that the condition depends on the depigmentation of the iris which is so common in myopes and also on a change in the corneal endothelium in the central area, which he imagines is also likely to occur in myopia owing to elongation of the eye.

S. R. GIFFORD.

Conjunctiva

PATHOLOGIC ANATOMY OF VERNAL CONJUNCTIVITIS. A. TORRES Y TORRES, Arch. de oftal. hispano-am. **34:** 126 (March) 1934.

Torres considers vernal conjunctivitis to be of a dermoidal nature. He found an increase in the thickness of the palpebral conjunctiva, with multiplication of the layers of the overlying epithelium, and considerable hypertrophy of the chorion with isolated formations and cellular infil-

tration. He did not find the reported eosinophilia as constant as is usually held. In some scrapings he found cellular inclusions such as have been reported in trachoma and in nongonococcic ophthalmia neonatorum. He compares the lesions described with those of follicular conjunctivitis, trachoma, phlyctenular infiltrations and Parinaud's conjunctivitis. He has never been able to find the bacillus described by Bellinzona and considers all the evidence as pointing to a nonbacterial etiology.

C. E. FINLAY.

Comparative Ophthalmology

THE RETINAL PIGMENT OF VERTEBRATES AND ITS RÔLE IN THE MECHANICS OF VISION. M. L. VERRIER, Compt. rend. Soc. de biol. **115**: 1078, 1934.

Among the vertebrates, pigmentation of the retina is not found in all species. Numerous elasmobranchs, for instance, have no pigment even in the portions of the retina most exposed to light. Among other fishes, the height of the pigmented epithelium varies from one half to one fifth of the thickness of the retina, and in reptiles attains only one twentieth of its thickness. No relation exists between the density of the pigment and the predominance of rods or cones. Among the pigment-lacking elasmobranchs, the torpedoes and sharks have only rods, while in the eagle-rays there is an alternation of cones with every two or three rods. Moreover, the deep sea fishes which possess only rods have as dense a pigmented epithelium as those fishes, such as the blennies, sea-horses and sea-bass, which have a developed fovea and cones in predominance. Similarly, although geckos, crocodiles and lizards all present the same type of pigmented epithelium the first possess only rods, the second rods and cones, and the last only cones. No relation, likewise, exists between the quantity of pigment and the illumination of habitat. The eagle-rays of the surface waters and the torpedoes of the deep show no variation in pigment, and the same observation is true in regard to diurnal and nocturnal reptiles and birds. Consequently both the importance of pigmented epithelium in the mechanism of vision and the protective rôle assigned to it are open to question.

J. E. LEBENSOHN.

THE NERVE ELEMENTS IN THE CORNEA OF THE GUINEA-PIG. I. EGOROW, Arch. f. Ophth. **131**: 531 (Feb.) 1934.

Since the work of A. S. Dogiel on the nerve endings in the cornea no similar studies have been made. Egorow therefore repeated the work with the same technic. Methylene blue (from 0.05 to 0.1 per cent) was used as a vital stain and ammonium molybdate as a fixative. Usually the whole cornea was examined directly (in toto); vertical sections embedded in paraffin or pyroxylin (celloidin) served only as controls. On the whole, Dogiel's classic observations could be confirmed. Typical encapsulated nerve endings were found in the transitional zone between the sclera and the cornea. "End-platelets" (Dogiel) and ramified endings were detected in the corneal stroma, and free "end-bodies" in the epithelium. The end-platelets and end-bodies had a fibrillary structure. Of special interest was the finding of the so-called

Eimer apparatus in the corneal epithelium. The Eimer apparatus in the muzzle of the mole was first described. The apparatus apparently represents a specific nerve end-body and consists of a number of epithelial cells which have become more intimately connected with the finest nerve fibers.

Egorow could also confirm the statement that nerve fibers enter epithelial cells. The great variability in the appearance of the peripheral parts of the neuron under normal conditions is stressed.

P. C. KRONFELD.

Congenital Anomalies

PERSISTENCE OF THE CIRCULATION IN THE POSTERIOR VASCULAR SHEATH OF THE LENS IN THE RIGHT EYE OF A GIRL AGED FOURTEEN YEARS. H. TOMKIN, Brit. J. Ophth. **18**: 39 (Jan.) 1934.

In the case which Tomkin reports a fine strand of pupillary membrane extended obliquely from the iris onto the anterior capsule of the lens. There were a few punctate opacities in the anterior cortex of the lens. Immediately behind the lens was an opacity in which vessels were radiating. It was thickest centrally, gradually thinning to the periphery, and occupied the middle third of the posterior part of the lens. The opacity was thin and irregular and had a serrated margin. Through the center a small opening could be seen, the point of entrance of the hyaloid artery, which divided into a descending branch and two nasal branches, the latter uniting again into a common trunk. With the slit lamp blood was seen coursing away from the center in all the branches. The lower column could be traced as far as the posterior surface of the iris. The common trunk could be traced a short distance and disappeared behind the iris. A number of fine gray strands were also seen, which had the appearance of obliterated vessels of the hyaloid artery. When the main trunk of the artery was traced backward with the ophthalmoscope, it appeared black, but a red glint along the upper border could be detected. The column was traced back to the disk. The hyaloid artery came from the central artery and was involved in connective tissue for part of its course from the disk. Color plates accompany the article. [A similar case has been reported by C. A. Young, of Roanoke, Va. W. Z.]

W. ZENTMAYER.

ALMOST COMPLETE ABSENCE OF THE MESODERMAL LAYER OF THE IRIS IN TWO GENERATIONS. OCULAR HYPERTENSION AND POLYCORIA IN ONE CASE. R. ROSSANO, Bull. Soc. d'opht. de Paris, January 1934, p. 8.

In the two cases presented, there existed a malformation of the cornea called "embryotoxin" by certain authors; the iris was reduced to its ectodermal layer covered by a bed of transparent stroma, containing most probably the dilator of the iris. The sphincter of the pupil was markedly visible, and the function was normal. In the first case, that of the father, there were two complete lacunae of the iris in the right eye, while the left eye showed hypertension. In the second case there were no lacunae and no hypertension, but the malformations of the corneas

and irides were similar to those in the other case. Accompanying the article are photographs of the irides, detailed reports of the patients, an adequate discussion of the literature and a bibliography.

L. L. MAYER.

Cornea and Sclera

CONICAL CORNEA COMPLICATED BY ACUTE ECTASIA. G. E. BERNER, Am. J. Ophth. **17**: 22 (Jan.) 1934.

Berner remarks the paucity of literature on acute ectasia complicating conical cornea. He reviews the literature and reports the case of a girl 14 years old who complained of sudden blurring of the vision of the left eye and a gray protuberance of the cornea. This occurred while she was playing basket-ball the previous evening. Her vision was: right eye, 6/60, left eye, 1/60. The right eye was normal except for a typical conical cornea. The left eye also showed a conical cornea, with the apex of the cone gray, opaque and bulging far forward, apparently on the point of rupture. A pressure bandage did not seem to halt this bulging. Repeated paracenteses were done, and the apex of the cone was touched daily with a solution of formaldehyde, (1:100) with eventual flattening of the bulging area. Each eye obtained vision of 6/9 with contact glasses.

W. S. REESE.

BILATERAL NECROSIS OF THE CORNEA FOLLOWING THE USE OF HAIR DYE ON THE EYEBROWS AND LASHES. C. T. MORAN, J. A. M. A. **102**: 286 (Jan. 27) 1934.

A woman aged 66 years applied Larieuse to the eyebrows and lashes. Both eyes began to itch and then to show redness and edema of the lids. One week later an ulcer of the cornea developed in the right eye and went on to perforation despite cauterization and intravenous injections of foreign protein and typhoid vaccine. Subsequently an ulcer of the cornea appeared in the left eye, and a conjunctival flap was drawn over it. Persistent increase of tension occurred, for which an iridectomy was done. A dermal test with the dye resulted in a 4 plus reaction. Six months after the onset of the ocular symptoms vision equaled 10/200.

Moran believes that removal of the lashes and eyebrows did more to alleviate the dermatitis than any other type of therapy, and that the conjunctival flap was a great factor in saving the left eye. If the sensitivity of the patient had been determined before the dye was applied the condition could have been avoided.

W. ZENTMAYER.

THE BIOMICROSCOPY OF THE NETWORK OF THE LIMBUS. A. ROLLIN, Arch. d'opht. **51**: 102 (Feb.) 1934.

Rollin emphasizes especially the biomicroscopic characteristics of the limbus which seem to play an important rôle physiologically and the study of which is worth consideration in certain pathologic conditions. The method of observation and the structure of the network are briefly described. The observation of the migration of pigment seems to indi-

cate not only that there is a current from the cornea and deeper structures toward the limbus but that the conjunctival circulation is in this direction. The limbus is consequently especially important in the absorption of drugs and other substances. From the point of view of disease Rollin deals only with the changes which are to be noted in acute, subacute and absolute glaucoma and in chronic glaucoma. In the former there is vasodilatation with stasis. The latter is characterized by the paucity of vessels in the limbus. This paucity of vessels is also seen in patients with chronic uveitis, those with atrophy of the globe and those with hypertension associated with arteriosclerosis. In hypotensive states the limbus is also exsanguinated. This report is concluded by a brief reference to the "palisades," which the author believes are undoubtedly lymph channels.

S. B. MARLOW.

THE ACTION OF A NEW PREPARATION OF VITAMIN A IN A CASE OF KERATOMALACIA. E. KUNZ, Klin. Monatsbl. f. Augenh. **92**: 74 (Jan.) 1934.

A girl aged 5 months was admitted to the hospital in a very neglected condition. Aside from keratomalacia, she suffered from bronchopneumonia of the right upper lobe, a papulous eczema of the legs, and rickets. A standardized preparation, containing 40,000 biologic units of vitamin A per cubic centimeter against from 345 to 690 units per cubic centimeter in other preparations, was given in doses of 5 drops three times a day, besides a proper diet and local ocular treatment. The weight increased, notwithstanding digestive complications, and the child recovered.

K. L. STOLL.

TREATMENT OF GROENOUW'S FAMILIAL DEGENERATION OF THE CORNEA. LOTTE HAGER, Klin. Monatsbl. f. Augenh. **92**: 116 (Jan.) 1934.

In two cases at the University Eye Clinic of Tübingen Groenouw's familial degeneration of the cornea was treated with Löwenstein's salve: ethylmorphine hydrochloride, U. S. P., 0.3 (to 0.6) cc.; mercurial ointment and petrolatum album, 5 Gm. each. One eye was bandaged alternately at night by the patients. In addition, from twelve to fifteen diathermic treatments were given, which may be repeated once or twice a year in case of necessity. Vision increased in one patient from 5/18 to 5/8 and in another from 5/6 to 5/5. Both patients were afflicted with the finely dotted type described by Groenouw.

K. L. STOLL.

A CASE OF SEVERE OCULAR SCROFULOSIS. W. STOCK, Klin. Monatsbl. f. Augenh. **92**: 117 (Jan.) 1934.

Very large phlyctenules recurred over and over again in both eyes of a girl aged 15. They involved nearly the entire conjunctiva bulbi, formed necrotic areas and left considerable scars. The cornea of the right eye was almost entirely scarred by large infiltrations and ulcers, reducing vision to mere perception of light. Owing to similar processes the vision of the left eye was reduced to 0.1/60. After all other treat-

ments failed, transfusion of 150 cc. of the mother's blood was resorted to. On the following day the eyes were less congested. No relapse occurred, complete recovery was obtained, the corneas cleared, and vision rose to 5/60. In the author's opinion certain blindness was prevented by the transfusion of blood.

K. L. STOLL.

Experimental Pathology

OCULAR EFFECTS OF CAROTID DECORTICATION (ARTERIAL SYMPATHECTOMY) IN MAN. MAGITOT and DESVIGNES, Bull. Soc. d'opht. de Paris, March 1934, p. 143.

A preliminary report of twelve observations of carotid decortication, both unilateral and bilateral, is made to show especially the effect of the operation on retinal arterial pressure, general arterial pressure, cerebrospinal pressure and intra-ocular tension.

In 1920 Leriche described the operation and noted the production of Horner's syndrome with dilatation of the conjunctival vessels. Bussy added the observation of a rise of intra-ocular tension of from 4 to 5 mm. on the side of the operation. These phenomena appeared in twenty-four hours and disappeared after some weeks. In 1930 Van Hoonacker tried this operation in the therapy of epilepsy. The operative procedure is outlined in detail. Magitot and Desvignes found on the side of the operation miosis, enophthalmos and decrease in size of the palpebral fissure, less than after removal of the superior cervical ganglion. Hyperemia of conjunctival vessels was not a constant finding. Epiphora was very inconstant. No change in the temperature of the face could be noted.

In the unilateral operation the retinal arterial tension was primarily lowered bilaterally, but this was transitory. Secondarily a hypertension ensued, reaching the maximum forty-eight hours after operation and remaining for from two to three days. A bilateral hypotension ensued in about eight days. Some days later the tension returned to normal.

In the bilateral operation, the second operation was done from ten to twenty-five days after the first. An immediate lowering of tension took place. Then hypertension occurred, lasting longer than in the unilateral cases. Hypotension then was noted but was not so marked as in the unilateral cases. Recovery to normal was quite prompt. The general arterial pressure after unilateral operation was unchanged, or raised at times from 5 to 10 mm. at the most and that was transitory. However, in the bilateral case there was an immediate rise of tension of from 30 to 40 mm. This lasted five days.

After bilateral sympathectomy the spinal pressure varied, and this was regarded as being due to variations in intracranial and intraspinal pressures.

Intra-ocular tension showed very slight changes. The authors present the study for its applicability in certain conditions of the optic nerve and retina.

L. L. MAYER.

Do Glucides Favor the Evolution of Experimental Xerophthalmia? G. Mouriquand and P. Chaix, Comp. rend. Soc. de biol. **115:** 538, 1934.

Bloch and Czerny found clinically in infants that a high proportion of glucides in a diet deficient in vitamin A tended to precipitate ocular lesions, especially xerophthalmia.

In experiments on animals, however, xerophthalmia appeared regularly on the third day of the avitaminic regimen independently of whether the diet was relatively rich or poor in glucides. J. E. Lebensohn.

Experimental Endocrine Cataract. G. Borsellino, Arch. di ottal. **41:** 56 (Jan.-Feb.) 1934.

Borsellino performed thyroidectomy and parathyroidectomy in rabbits and describes his technic. None of the thyroidectomized animals acquired lens opacities. In two of the five parathyroidectomized animals opacities developed after a month. They consisted in fine white flecks in the anterior and posterior cortex and along the posterior suture lines. One animal showed a denser oval one in the posterior cortex. These opacities became stationary and remained so during an observation period of five months. Tetany did not develop in any animal, owing, no doubt, to the presence of accessory parathyroid tissue.

S. R. Gifford.

Adhesive Chorioretinitis Produced by Diathermic Coagulation. C. Cordero, Arch. di ottal. **41:** 65 (Jan.-Feb.) 1934.

Rabbits' eyes were treated with various doses of electrocoagulation and examined histologically. After perforating coagulation in which from 150 to 200 ma. was used for from two-tenths to three-tenths second [!] the reaction was moderate and new connective tissue was seen after from eight to ten days. After from sixty to seventy days the canal made by the coagulating needle was filled, and adhesion was present between the retina and the choroid at the edges of the canal. After coagulation of the scleral surface a severe reaction of the whole eye was produced; the sclera became necrotic, and the retina was destroyed in the corresponding area. Hence the method of perforating coagulation seems much the safest in the treatment of retinal detachment.

S. R. Gifford.

The Ocular Lesions Produced by Naphthalene. A. García Miranda, Arch. de oftal. hispano-am. **34:** 76 (Feb.) 1934.

After an extensive review of the literature and theories in relation to ocular lesions produced by naphthalene and reference to his own experiments on rabbits, to the clinical features of these, to local and general metabolic changes, to the lesions and to slit-lamp studies, García concludes that the ocular lesions cannot be attributed to metabolic disturbance, and that naphthalene cataract always follows retinochoroidal lesions and must therefore be considered as a complicated cataract.

C. E. Finlay.

THE INFLUENCE OF LIGHT OF GREAT INTENSITY ON RABBIT EYES. D. SILBE, Arch. f. Ophth. **131: 586** (Feb.) 1934.

The purpose of the experiments was to determine the maximum amount of visible rays which is tolerated by the eyes of the rabbit without pathologic changes in the retina or the lens. The method was the one used by Vogt and his associates, slightly modified to make the experimental conditions more closely comparable with those prevailing in factories where men expose their eyes to light of great intensity. Strong electric bulbs and an arc lamp, both with reflectors, were used as sources of light. Heat rays were eliminated more or less completely by a filter consisting of a copper sulphate solution in a glass container. After passing the filter the light rays were concentrated on the eye of the rabbit by means of a hollow sheet-metal cone. If the heat rays were not entirely eliminated the eye responded with mild conjunctivitis and desquamation of the corneal epithelium. The visible rays produced no noticeable changes in the retina or the lens even when the unfiltered light of an arc lamp was thrown by means of a parabolic mirror into the eye for fifty-five hours. It seems doubtful, therefore, that visible rays of any intensity can damage ocular tissues to any degree detectable by ordinary histologic examination.

P. C. KRONFELD.

EXPERIMENTS ON RABBITS WITH BIPOLAR SURFACE ELECTROLYSIS IN THE TREATMENT OF DETACHMENT OF THE RETINA. A. VON SZILY and H. MACHEMER, Klin. Monatsbl. f. Augenh. **92**: 44 (Jan.) 1934.

Experiments with diathermy applied carefully and with low currents of from 50 to 100 milliamperes were followed occasionally by degenerations, hemorrhages, tears and detachments of the retina. The galvanic current appeared to be applicable with less difficulty and greater accuracy. Von Szily and Machemer experimented with the galvanic current, using both poles as active electrodes. They placed the electrodes on the eye a short distance apart, producing a current between them only. Following Engelking's method they used this current for surface treatment and for perforation. Weak galvanic currents were passed through a small area of the coats of the eye by means of a bipolar instrument placed on the sclera. Correct dosage of both electrodes produced an inflammation which resulted in reattachment of the retina. The instrument and its application are described, and the results are illustrated by a number of microscopic and ophthalmoscopic pictures. Equal results obtained by equal doses are evident. Slight and also very intensive coagulations may be produced according to the power of the current, the duration of the application, the thickness of the needle and the distance between the two poles. In forty rabbits' eyes currents of from 2 to 20 milliamperes and applications of from 5 to 20 seconds produced no tears outside the treated area and only exceptional minute hemorrhages from the retinal scar. Opacities of the vitreous were observed only after the use of high currents. Retinal degenerations rarely extended beyond the treated area. Complete degeneration of the retinal elements within the reattached area occurred rarely.

K. L. STOLL.

General Diseases

TWO CASES OF OBLITERATION OF THE CHOROIDAL NETWORK, ONE PATIENT HAVING RAYNAUD'S DISEASE. BAILLIART, H. TILLÉ and M. LAIGNIER, Bull. Soc. d'opht. de Paris, January 1934, p. 17.

In two patients lesions of the retinas and the symptoms were secondary to an obliteration of the vascular tree of the choroid. It is impossible to give a definite cause for the condition. The authors believe that Raynaud's disease and angina pectoris, which were the general diagnoses in these cases, may have some direct relation to the choroidal vascular changes. The value of being able to view these lesions with the ophthalmoscope is emphasized.

L. L. MAYER.

RAYNAUD'S DISEASE WITH PERIARTERITIS RETINAE. JEAN-GALLOIS and DANIEL ROUTIER, Bull. Soc. d'opht. de Paris, January 1934, p. 32.

The authors report a case in which there was much evidence of generalized arterial spasm. The evidence given leaves no question as to the diagnosis of Raynaud's disease. In addition they were able to observe in one eye of the patient a tendency to periodic arterial spasm. Other vessels in this same eye showed marked periarteritis, which the authors construe as being the result of intermittent traumatism to the wall of the vessel by the periodic spasm of the musculature. These observations are presented so that others may be on the lookout for such evidence in Raynaud's disease.

L. L. MAYER.

RARE OCULAR CHANGES IN SCHÜLLER-CHRISTIAN DISEASE: REPORT OF A CASE. P. A. JAENSCH, Klin. Monatsbl. f. Augenh. **92**: 158 (Feb.) 1934.

Jaensch describes some rare ocular changes in a boy, aged 10, with all the symptoms of Schüller-Christian disease: defects in the skull, exophthalmos and diabetes insipidus. Numerous small xanthomas in the epidermis, especially of the lids, were found; furthermore, there were lipoid granulomas of the conjunctiva and the limbus corneae and a similar formation on the disk. Partial fatty degeneration, similar to interstitial keratitis, seen in the cornea, was diagnosed as a primary deposit of lipoid. The formation at the limbus, with a diameter of 6 mm., did not involve the cornea; it was interpreted as a granuloma with a secondary deposit of lipoid, analogous to that in Ceelen's case.

Considerable changes took place in the course of six months: The fatty degeneration of the cornea receded, while the individual spots in the cornea grew larger, assuming a silvery-white hue. Small dark-brown spots developed in the macula, which had been free from hemorrhages and other changes hitherto; the formation in the disk remained unaltered, but for slight periodical variations of its thickness. The Wassermann and Pirquet reactions were negative.

K. L. STOLL.

Glaucoma

SUPPLEMENTARY NOTE ON IRIS INCLUSION FOR CHRONIC GLAUCOMA. H. HERBERT, Brit. J. Ophth. **18:** 142 (March) 1934.

In a previous paper (*Brit. J. Ophth.* **14:** 433, 1930) Herbert developed the thesis that progress in iridencleisis has for more than a decade resolved itself into a practical study of conjunctival fibrosis and protection against late infection. There have been three phases in this experience. In the final very short period a definite increase in fibrosis has been secured, without any restriction of the required subconjunctival filtration, by the addition of a measured iridodialysis, which is obtained by seizing the iris near its base and making a steady pull of the iris forceps toward the center of the pupil. The detachment should have an extent of from 3 to 4 mm. There is some reason to hope that the early increased fibrosis secured by the addition of iridodialysis will be permanent.

The only postoperative case of sympathetic ophthalmitis that has come under the author's observation in England occurred in a case of ordinary combined extraction of cataract. While he attributes the condition to excessive and ill-timed use of antiseptic solution, he holds that this should not be allowed to detract from the undoubted value of its correct employment.

W. ZENTMAYER.

RELATION BETWEEN THE POTASSIUM AND CALCIUM CONTENTS OF THE BLOOD OF PATIENTS WITH GLAUCOMA. A. BIFFIS, Klin. Monatsbl. f. Augenh. **92:** 138 (Jan.) 1934.

Biffis examined the blood of fifty patients with glaucoma and that of twenty healthy persons as controls. He found in the blood serum of those with glaucoma, on the average, a slight increase of potassium and a decrease of calcium, i. e., a slightly higher potassium-calcium quotient than in healthy persons. An increased tonus of the parasympathetic and slight alkalinization seem to exist in primary glaucoma.

K. L. STOLL.

Hygiene, Sociology, Education and History

DATA CONCERNING RADIATION AND PROTECTION GLASSES WITH A NOTE ON RETINOSCOPES. A. RUGG-GUNN, Brit. J. Ophth. **18:** 65 (Feb.) 1934.

Popular taste in regard to the proper enjoyment of leisure has developed along lines that call for, or appear to call for, some measure of protection for the eyes. The object of the paper is to assemble the data relating to radiation, and to attempt to analyze the factors against which protection is desirable and the means at one's disposal for securing it. The summary of the related data regarding radiation is a follows: . . . "at the poles, in the tropics, at sea-level and at altitudes up to 4,000 ft., the range of the ultra-violet spectrum remains constant, extending to a wave-length of about 2,900 Å. The intensity of radiation in the ultra-violet region, on the other hand, is increased through selective dispersion and scattering by ionized particles—indirect

blue sky radiation; through non-selective reflection by light clouds and atmospheric haze; and by non-selective reflection from the surface of the earth, particularly from snow and ice-fields, water and desert. The relative absence of suspended dust at altitudes of 6,000 ft. and over permits a high ultra-violet intensity in winter as well as in summer. Tropical sunlight is also characterized by high ultra-violet intensity constant throughout the year."

The related data as to infra-red rays are summarized as follows: . . . "the near infra-red region, from 7,500 Å to 9,500 Å is characterized by the property of haze penetration. From 9,000 Å onward strong water-vapour absorption takes place. High infra-red intensity is associated with the decreased humidity of mountain summits and low intensity with areas near the sea even in tropics. This is at a maximum where the soil is barren of vegetation but occurs also from grassland, crops and forests. Infra-red intensity is insignificant over snow or ice-fields or surfaces of water."

The related data as to visible rays are summarized as follows: "Glare, in short, is largely the effect of polarized light and is present whenever the eye is so placed as to receive reflected rays at the polarizing angle. More accurately, glare, as represented by specular reflection, is a resultant of two components; (1) polarized light which is present whenever the eye is so placed as to receive reflected rays at the polarizing angle; and (2) ordinary regular reflection combined with a certain amount of diffuse reflection in all other relations of the eye to the reflected light. The intensity of the former, which is by far the more important, can be diminished only by some such instrument as a plate of tourmaline acting as an analyser; the latter perhaps only by selectively absorbing or reflecting the rays in the region of maximum energy; *i. e.*, in the yellow-green round about wave-length 5,500 Å."

A brief description of the composition of glasses is given, followed by a discussion of the practical application of the data. The term "colored" appears to be preferable to "tinted." A tinted glass is a glass feebly saturated with color, a description that is applicable to only a few varieties of protective glasses. Protection against glare can be achieved for all practical purposes by wearing lenses of Chance's didymium glass. Crookes A.1 also contains didymium. For more serious cases of intolerance there are available the members of the Crookes series: A.2, B.1 and B.2. For aviation, Crookes B.1 or B.2 with a thin metallic film adherent to its outer surface is theoretically the ideal glass. The objection is that the film wears off or becomes detached. There is now being perfected, however, a process that promises to solve these difficulties. For conditions which obtain in arctic and antarctic snow-fields laminated glass is advisable, such as Crookes B.2 on which a metallic film, e. g., gold, has been sputtered.

Regarding frames it is pointed out that when goggles are worn in the polar latitudes the metal should not come in contact with the skin.

In a note on retinoscopes the author states that reflecting glass retinoscopes are particularly suitable for minor errors but are not suitable for high myopes or for eyes in which opacities are present in the lens or the cornea.

W. ZENTMAYER.

AUTOMOBILE ACCIDENTS AND HEMERALOPIA. J. ROLLET, Arch. d'opht. **51:** 72 (Feb.) 1934.

Rollet gives an account of two automobile drivers who had met with repeated accidents at night and finally with two fatalities. Both drivers were found to be suffering from hemeralopia without fundus changes. He points out that police statistics show that the greatest number of automobile accidents occur at nightfall, so that it is probable that lowered visual acuity in dull light plays a not negligible part. For this reason he thinks that it should be compulsory for those having to do with public carriers and those who have had repeated accidents to be tested out with lowered illumination, even if it is impracticable to test all drivers. He also suggests that this procedure be used in examinations of applicants for employment on railroads and in aviation.

S. B. MARLOW.

Injuries

OCULAR DISTURBANCES FOLLOWING COMPRESSION OF A FRONTAL HEMATOMA. E. DELORD, Arch. d'opht. **50:** 565 (Aug.) 1933.

Delord reports a case in which compression of a frontal hematoma, which was painful and had persisted for a month, was followed by optic neuritis. The hematoma followed an automobile accident. Compression of this hematoma was followed first by extensive edema of the lids and malar region and then by conjunctival edema. This disappeared but was followed by edema of the optic nerve, reduction of vision to 1/20 and pain on movement of the globes. Spontaneous cure occurred four days later.

S. B. MARLOW.

Lacrimal Apparatus

CHRONIC CONJUNCTIVITIS CAUSED BY UNRECOGNIZED CONCRETIONS OF THE LOWER LACRIMAL CANALICULUS. VELIÈRE-VIALEIX, Ann. d'ocul. **170:** 1030 (Dec.) 1933.

The classic symptoms found were epiphora and tumefaction of the inferior canaliculus. The secretion, mucopurulent and purulent, was abundant, and the lacrimal signs were often absent or appeared late. One should immediately think of a canalicular concretion in the presence of a unilateral conjunctivitis that resists local treatment. Even though there are no lacrimal symptoms in such a case, the canals should be incised and explored with a curet. It is also well to have a microscopic examination of the secretion made for identification of Streptothrix försteri, if present.

S. H. MCKEE.

ARGYROSIS OF THE LACRIMAL SAC. C. A. GABRIÉLIDÈS, Ann. d'ocul. **171:** 232 (March) 1934.

The character and the use of silver salts and their manifestations in the skin and mucous membranes have been known for a considerable period from studies by chemists, physiologists and ophthalmologists.

The pigmentary manifestations of the silver salts in the eye are frequent enough through argyrosis of the conjunctiva and cornea. Argyrosis of the lacrimal sac is probably not so rare as is thought, since an anatomic-pathologic examination is often needed to verify it. Dupuy-Dutemps stated that argyrosis of the lacrimal sac is not exceptional, and usually accompanies argyrosis of the conjunctiva. Many agree with this view.

The case reported is one of purulent dacryocystitis in a patient with argyrosis who was operated on for the dacryocystitis. The details of the case are given, also the histologic report, in which it is stated that the changes in argyrosis of the lacrimal sac are identical with those of argyrosis of the skin due to precipitation of silver. S. H. McKee.

Action of Pilocarpine, Atropine, Extract of Lacrimal Gland and the Oculocardiac Reflex on the Sodium Chloride Content of Tears. D. Michail and P. Vancea, Comp. rend. Soc. de biol. **115:** 1703, 1934.

The salt content of tears tends to decrease after instillations of pilocarpine into the conjunctival sac, and to increase after instillations of atropine or extract of lacrimal gland. The oculocardiac reflex has a diphasic effect, inducing a primary decrease followed by a secondary increase of the sodium chloride content. J. E. Lebensohn.

Action of Retrobulbar and Subcutaneous Injections of Salt Solutions on the Salt Content of Tears. D. Michail, Pacurariu and Zolog, Comp. rend. Soc. de biol. **115:** 1706, 1934.

Retrobulbar injection of 1 cc. of 2 per cent sodium chloride through the lower lid tends to increase the salt content of tears, probably owing to regional neurovegetative stimulation. Physiologic solution of sodium chloride injected into the temples may be followed by either an increased or a decreased concentration of salt in subsequent tears.

J. E. Lebensohn.

Lens

Cataract as a Result of Dietary Deficiency in Larval Amblystoma Tigrinum. E. M. Patch, Science **79:** 57 (Jan. 19) 1934.

The author's abstract is as follows: "In larvae of the tiger salamander being reared on a series of highly purified milk-powder-casein diets, the lenses of the eyes turned milky-white in mid-larval life and passed through stages like those of "senile" cataract of the cortical type. At the stage of swelling there appeared a total opacity of the lens cortex with degeneration of the cortical fibers, but the lens nucleus retained its crystalline transparency. It was clear that inadequacy of the basic milk diet had produced the disease, because it was lacking in other larvae from the same egg-masses fed on a series of synthetic beef muscle diets. Ten animals from the milk-fed groups had visible cata-

racts when changes were made in the rations. Increase of vitamin A or of vitamin G was ineffective in preventing development of the disease; nine new cases of the eye defect appeared. In another group of larvae fed on one of the milk-powder-casein diets, one case of cataract was developing when dietary addition was given. With purified cystine as food supplement and no change in the vitamin supply, the other members of this group remained free from the eye opacity."

F. H. ADLER.

SLIT-LAMP FINDINGS IN THE EYES OF STOKERS DURING AND IMMEDIATELY AFTER THEIR WORK. W. F. SCHNYDER, Arch. f. Ophth. **131:** 599 (Feb.) 1934.

This paper represents a continuation of the controversy between Goldmann and Vogt and his associates about the cause of fire cataract. Schnyder examined ten stokers immediately after they had been exposed to the radiation of melted iron. He found signs of overheating of the anterior segment. The experiments in which Goldmann claimed to have produced fire cataract by conduction of heat from the iris are interpreted differently by Schnyder. He thinks that Goldmann burned the iris repeatedly and thus produced recurrent iritis with subsequent atrophy and hole formation. That the cataract which occurs under those conditions is of the complicated type Schnyder tries to prove by its similarity to other well known types of complicated cataract.

P. C. KRONFELD.

THE BODY TEMPERATURE OF STOKERS. W. F. SCHNYDER, Arch. f. Ophth. **131:** 611 (Feb.) 1934.

The body temperature of stokers actually increases during their work at the furnace. The average increase in ten laborers was 0.6 C., not 1.35 as claimed by Goldmann. According to Schnyder there is no reason to consider a slight increase in body temperature as a possible noxious agent in connection with the problem of fire cataract.

P. C. KRONFELD.

Lids

AUTOVACCINE IN ULCEROUS BLEPHARITIS. K. V. SNEGIREFF, Sovet. vestnik oftal. **4:** 208, 1934.

During the past nine years Snegireff has used an autogenous vaccine in 120 patients (210 eyes) suffering from ulcerous blepharitis which had not responded satisfactorily to the routine local and general treatment. The ages of the patients varied from 5 to 45 years and the duration of the disease from 1 to 25 years. In all but 6 patients Staphylococcus aureus was found on bacteriologic examination. The injections, numbering from 8 to 16, were given subcutaneously into the lids, temple or arm. In 32 per cent a complete cure was obtained, in 55 per cent marked improvement followed, and in 13 per cent there was very little improvement.

O. SITCHEVSKA.

Methods of Examination

A Transparent Protractor for Scotometers. J. Foster, Brit. J. Ophth. **18:** 98 (Feb.) 1934.

After the scotometric map of the central field of vision has been made a protractor of heavy transparent cellophane, marked with radial lines and concentric circles, is unrolled from a roller-blind mounting above the screen or is pinned to the screen through four eyelet holes, so that the center of the protractor is over the fixation point, and the upper and lower margins are horizontal. The transfer of the map, outlined by the pins on the screen, to the chart is then made.

W. Zentmayer.

Synchisis Scintillans, Cinematographic and Stereophotographic. Lijo-Pavia, Bull. Soc. d'ophth. de Paris, January 1934, p. 35.

Synchisis scintillans is described as seen in the stereoscopic moving picture. The author is able to confirm Koby's vivid description of this phenomenon as observed with the slit lamp. Certain characteristics of the opacities are well noted in the moving picture, viz., the disposition in the form of a funnel, their magnitude and the rapidity of their movement.

L. L. Mayer.

Gonioscopy. A. Trantas, Arch. di ottal. **41:** 39 (Jan.-Feb.) 1934.

Trantas describes his method of examining the angle of the anterior chamber and the ciliary region by making digital pressure on the sclera while using the ophthalmoscope. This was described in 1899 and was the first attempt at careful examination of this angle. It is varied by transilluminating the sclera while making pressure, the observation being made with the corneal microscope. He describes the canal of Schlemm as seen in normal eyes and in those with glaucoma. The canal is normally empty of blood but was seen filled with blood during acute hypotony in an eye with retinal detachment. In high myopia a hyperpigmentation of the angle was common (ten of twenty-two eyes). This was also true in retinal detachment (nine of fourteen eyes).

In glaucoma the angle was normally open in 47 per cent of thirty-eight cases. In 29 per cent it was only partly visible and in 24 per cent it was invisible, though even in these cases it was not closed completely.

The posterior surface of the cornea could be observed in areas where it was opaque, by observation through a clear area at the opposite side of the eye.

S. R. Gifford.

Neurology

The Participation of the Facial Nerve in the Motor Innervation of the Lids: The Superior Facial. E. Velter, Arch. d'opht. **51:** 65 (Feb.) 1934.

The motor involvements in facial paralysis long ago made apparent the distinction of the kind and the localization of the lesions which produce them. On the one hand are the peripheral lesions which

involve all the muscles supplied by the facial nerve and on the other the central lesions which have to do with the lids, the brow and the forehead. In addition to these clinical facts there are certain physiologic observations which show that there is a synergistication with the oculomotor nerve. These clinical and physiologic phenomena have led neurologists to conceive of an autonomy of the facial nerve. Velter presents and discusses the physiologic and clinical observations which are known. He briefly outlines the two hypotheses which have been proposed to explain them: (1) the existence of a special center for the superior facial nerve in the nucleus of the seventh nerve and (2) the existence of a separate cortical center with special corticonuclear connections. The evidence in support of these two hypotheses is discussed with reference to the literature. He expresses the opinion as a result of these investigations that it is possible to maintain on clinical and physiologic grounds that there is a relative autonomy of the superior facial nerve. This, however, is not dependent on a special cortical center but is brought about by bilateral corticonuclear connections through the geniculate bundle on the one hand (the pyramidal tract) and, on the other, sufficient pliancy of the integrity of the latter to permit physiologic synergy with the muscles supplied by the superior facial nerve. S. B. MARLOW.

CLINICAL OBSERVATIONS IN HEMICRANIA VASOMOTORICA WITH DISTURBANCES OF THE VISUAL ORGAN: REPORT OF CASES. F. POOS, Klin. Monatsbl. f. Augenh. **92**: 58 (Jan.) 1934.

Poos reports eight cases of vasomotor migraine complicated with uncommon disturbances of the visual organ.

1. A man had typical periodic migraine with scintillating scotomas from the thirteenth to the thirty-fifth year of his life. At the age of 53 permanent complete homonymous hemianopia on the right side with preservation of the macula developed without return of the migraine. The hemianopia was observed for three years.

2. Transient paralysis of the abducens muscle of the right side occurred in a girl, aged 18, during an interval of migraine without an attack. During the following days the vascular spasms extended to other cerebral centers (so-called migrating vascular crises); they produced paresthesias and pareses in the extremities of the same side. Complete recovery followed within from three to four months independently of new attacks of migraine and scintillating scotomas.

3. Recurring paralysis of the abducens and inferior rectus muscles was observed in a man, aged 48, who had suffered from migraine for eighteen years.

4 and 5. Two cases of recurring paresis of the trochlear muscle, the one of the left eye, the other of the right, were observed, respectively, in a woman, aged 30, and a girl, aged 21. In the first patient application of an ice-cap to the head was promptly followed by double vision and headache. The same exacerbation of the symptoms of migraine could be brought on in this manner in case 3.

6 and 7. Two cases of typical recurring paresis of the oculomotor muscle were observed in two men, aged 43 and 29, respectively. They presented the peculiarities, symptoms and clinical course typical of Charcot's ophthalmoplegic migraine.

8 and 9. Two patients had migraine, a girl, aged 12, and a woman, aged 44. In the girl the condition was complicated by isolated dysfunction of the pupils, anisocoria, and by Argyll Robertson pupil in the right eye; in the woman, by anisocoria with Argyll Robertson pupil in the right eye and iridoplegia in the left eye. The first case was one of an allergic form of juvenile migraine with predominant abdominal symptoms. The second case was one of juvenile periodic migraine in which the ocular symptoms developed during the menopause.

In closing his article Poos discusses the treatment of migraine. He emphasizes the importance of finding and treating the causative factor, e. g., its endocrine origin. Suggestions are given relative to refraction, diet, rest, change of climate, treatment with diathermy and other heat therapies. Applications of cold and the use of alcohol, nicotine, caffeine and certain drugs are contraindicated.

K. L. STOLL.

Ocular Muscles

THE LOCALIZATION OF THE APPARENTLY VERTICAL IN MOTOR DISTURBANCES OF THE EYE. W. HERZAN, Arch. f. Ophth. **132**: 101 (March) 1934.

The apparently vertical or horizontal direction is chiefly determined by the spatial values of the retinal elements if the test is done in such a way that labyrinthine and proprioceptive impulses are excluded as much as possible and if the person under examination cannot correlate the target line with any known directions in the room. The position of the apparently vertical line in normal subjects is known to vary between approximately 1 degree of conclination and 3 degrees of disclination. Greater differences between the objective and the subjective vertical are rare.

Herzan, an associate of Bielschowsky, determined the position of the apparently vertical in the dark and in the light in a large number of ocular palsies in which muscles with torsional components were involved. In addition, the degree of pathologic cyclotropia (*Verrollung,* extorsion or intorsion) was determined by letting the patient fuse in a stereoscope two movable radii. The main result was that the apparently vertical is almost always tilted in the same direction and to the same degree as the principal longitudinal section of the retina. Under the conditions of the test, namely, monocular fixation with head in primary position and exclusion of empirical factors, the absolute localization of directions remains depending on the original spatial values.

The determination of the apparently vertical can be a valuable diagnostic test in cases in which a paralysis of one or more elevators or depressors has developed into a concomitant squint and also in cases in which motor disturbances must be diagnosed although the patient has only one seeing eye.

P. C. KRONFELD.

EXOPHORIA AND "EXCESS DIVERGENCE": REPORT OF A CASE. A. BIELSCHOWSKY, Klin. Monatsbl. f. Augenh. **92**: 11 (Jan.) 1934.

Bielschowsky renders a detailed clinical and therapeutic account of a case of disturbed fusion of organic and psychogenic nature. He discusses the question whether certain forms of exophoria may be caused by an excess of divergence, as stated by Duane.

A girl, aged 22, noticed failing vision after an attack of grip; spasm of accommodation and crossed double images were also noted. Atropinization, bilateral advancement of the internal recti and later on partial central tenotomy of both abducens muscles were of no avail. After eleven years' observation and treatment the final condition was this: The patient had occasional diplopia at a distance and depending on her general condition, a varying degree of insufficiency of convergence evident by crossed double images at a close range. An attempt to fuse the images was followed by a spasm of accommodation and convergence, which did not produce an increase of the refraction, owing to continued use of atropine, but during which the crossed diplopia changed to homonymous diplopia. Paresis of the muscles of convergence may be ruled out, because the patient could obtain maximal convergence for certain periods of time. The patient had the choice between two evils: either to use atropine and to see plainly with proper glasses while she was disturbed by crossed double images, or to omit atropine and facilitate her work at a close range, but risk more severe and more frequent attacks of accommodative spasm and an increase in her myopia. Occlusion of one eye being impossible owing to her work at the postoffice, Bielschowsky considers eventual use, for a close range, of strong abducing prisms mounted on the lower portion of the spectacles.

The source of the disturbance was exophoria, which remained latent until the patient's resistive power was weakened by grip with spasms of accommodation resulting. Disagreeing with Duane in some particulars, Bielschowsky, after observing this unique case, has laid aside his skepticism regarding the existence of excesses of divergence. The disturbance is rare and may be related to the "spasms of the externi muscles" in hysteria. Symptoms, course and therapeutic results in the case described suggest the presence of an abnormal irritability of the nervous mechanism of divergence. It is probable that exophoria, i. e., an anomaly of the anatomic balance, existed originally. This anomaly was absent after the bilateral advancement of the internal recti. In closing, he discusses the merits of correction of, and exercise with, prisms and arrives at the conclusion that advancement or shortening of both internal recti is the operation of choice in all exophorias.

K. L. Stoll.

Operations

Extensive Oculo-Palpebral Neoplasm; Excision of Lids and Enucleation of the Eyeball Followed by Occlusion of the Orbit. E. Puscariu, Brit. J. Ophth. **18:** 101 (Feb.) 1934.

Puscariu's procedure is for the removal of a neoplasm of the bulb and lids involving the orbit, whether the tumor has in the beginning an epibulbar or, as happens more frequently, a palpebral origin.

The steps of the operation are as follows: First, four linear incisions are made tangent to the four orbital borders, uniting with one another at right angles. Each incision involves the skin and subjacent muscles. Second, an incision of the orbital aponeurosis is made, revealing the girth of the superior rectus muscle, which is divided. The same process is repeated for the three other incisions, separating the aponeurosis from its orbital insertions, cutting the other three muscles and sectioning

also the capsule of Tenon, which extends between them. After the orbicular tendon is detached, and seized with a hemostatic forceps, the internal rectus is divided. The third step is the luxation of the eyeball and the section of the optic nerve and of the last muscles and adhesions. To cover in the area, two cutaneous flaps are cut, one frontal and the other facial, the vertical incisions being extended to the necessary lengths. The flaps, well dissected, are advanced one toward the other in the face of the orbit and are sutured about its center. An illustrative case is given, and the steps of the operation are figured.

W. ZENTMAYER.

THE USE OF THE READY-MADE CARTILAGE IMPLANT AFTER ENUCLEATION. A. LOEWENSTEIN, Klin. Monatsbl. f. Augenh. **92**: 86 (Jan.) **1934.**

Loewenstein postulates that in the use of an implant there is slow absorption with consequent formation of a resistive hull. He discusses the merits of various materials and describes the preparation and implantation of a sterile implant of cartilage. The method of its preparation is analogous to the manufacture of catgut. It contains a small piece of bone and need not be entirely covered by periosteum or perichondrium. Pierced by two double-armed catgut sutures it can be fixed within Tenon's capsule in a manner which will prevent rotation. The spheroid implants are furnished in sealed glass tubes, and each is pierced with two double-armed sutures, ready for a tobacco-pouch closure of the conjunctiva. The implants have a diameter of from 15 to 20 mm. Eight of nine patients operated on at the clinic as outpatients obtained satisfactory cosmetic results and received the prosthesis from four to five weeks after the operation.

K. L. STOLL.

Pharmacology

THE INFLUENCE OF p_H ON THE STABILITY OF COCAINE HYDROCHLORIDE SOLUTIONS. J. REGNIER and R. DAVID, Compt. rend. Soc. de biol. **151**: 1195, 1934.

The best buffer solution in which to preserve cocaine hydrochloride is one of acetic acid and sodium acetate at p_H 4.2. In this buffer the cocaine is unaffected by age or sterilization. Phosphate buffers of the same p_H are not similarly effective.

J. E. LEBENSOHN.

EXPERIMENTAL AND CLINICAL RESEARCHES ON THE USE OF OXYQUINOLINE SULPHATE IN OPHTHALMIC CONDITIONS. H. BRACCI-TORSI, Ann. di ottal. **61**: 775 (Oct.) 1933.

Powerful bactericidal properties have been ascribed to the sulphate of ortho-oxyquinoline, a nonirritating and soluble derivative of quinoline (C_9H_7N), by previous workers in other branches of medicine. In a concentration of 1 : 500 it killed cultures of many pathogenic organisms in twenty-four hours. In the presence of serum, pneumococci were killed after five minutes by a concentration of 1 : 1,000. Two strains of Streptococcus were killed by a 1 : 5,000 solution, a third strain requiring a concentration of 1 : 1,000. A strain of Staphylococcus aureus was

killed by a 1:5,000 solution in five minutes. Bacillus diphtheriae, Bacillus coli and Bacillus pyocyaneus were resistant even to a 1 per cent solution. Subcutaneous injections into rabbits immunized to Bacillus typhosus and Brucella melitensis produced a marked increase in specific agglutinins. A 1 per cent solution used for instillation in rabbits caused no damage to the epithelium and did not interfere with the healing of artificial erosions. In rabbits 0.2 cc. of a 1 per cent solution injected into the anterior chamber produced a slight iritic reaction which cleared up completely in three days.

Eleven patients with corneal ulcer were treated by instillation of a 0.5 per cent solution twice a day. Only moderate burning was complained of. Most of the cases were due to pneumococci, and many were accompanied by hypopyon. In most of the cases the process appeared favorably affected by the drug, though paracentesis was necessary in several.

S. R. GIFFORD.

THE ACTION OF ACETYLCHOLINE IN QUININE AMBLYOPIA AND AMAUROSIS. L. MAZZI, Arch. di ottal. **41:** 27 (Jan.-Feb.) 1934.

Mazzi concluded from previous work that the visual loss in quinine poisoning is due to a direct effect on the retinal cells and not to vasoconstriction. In a new series of two dogs he produced amaurosis or extreme amblyopia by injecting from 32 to 37 cg. of quinine hydrochloride per kilogram subcutaneously. One eye was enucleated after ten days, whereupon daily injections of acetylcholine were begun, and the other eye was enucleated after a week. There was no apparent effect on the animal's vision during life nor was any difference seen between the eyes enucleated before and after the use of acetylcholine.

Reports of the favorable effect of acetylcholine given early in various conditions may probably be explained by its vasodilator action, the resulting hyperemia removing the toxin more rapidly from the retina and aiding the damaged retinal cells to recuperate. S. R. GIFFORD.

Physiology

A CONTRIBUTION TO THE KNOWLEDGE OF THE CIRCULATION OF THE AQUEOUS. A. VON PFLUGK, Arch. f. Ophth. **131:** 614 (Feb.) 1934.

Von Pflugk has succeeded in taking moving pictures (twenty-four per second) of pupillary reactions. He describes in detail his photographic observations of a filament of a persistent pupillary membrane which ran almost straight across the upper portion of the pupil. When the pupil contracted the filament became curved with its convexity pointing up and forward. In his own words "the filament was thrown upward and toward the cornea." This movement of the filament against the influence of gravity indicates the presence of a current in the aqueous from the pupil toward the cornea. This current is produced by the contracting iris displacing the aqueous which is present in the pupillary area. The author assumes further that through the sudden change in the amount of light entering the eye the ciliary processes are somehow put under pressure so that they produce more fluid, making the outflow from the pupil more pronounced. P. C. KRONFELD.

Retina and Optic Nerve

RETINAL DETACHMENT. C. B. WALKER, Am. J. Ophth. **17:** 1 (Jan.) 1934.

Walker states that Verhoeff was the first to use multiple small needles inserted through the sclera to carry a cauterizing electrolytic current to the choroid to stimulate an adhesive choroiditis. This article is too technical and discursive to abstract. He describes his own method of curing retinal detachment by diathermy and includes an illustrative case. He concludes that: 1. Diathermy equipment is now necessary to the ophthalmologist. 2. Diathermic micropuncture will probably be perfected so as to supplant other methods of treating retinal detachment. 3. Single, noninsulated, cleanable pins with very small stops are an advantage. These pins should be made of iridium-hardened platinum and attached to threads.

W. S. REESE.

THE CLINICAL SIGNIFICANCE OF RETROBULBAR AND OPTIC NEURITIS. W. I. LILLIE, Am. J. Ophth. **17:** 110 (Feb.) 1934.

Lillie discusses retrobulbar and optic neuritis, and stresses the necessity for thorough general and neurologic examinations in these conditions. Only by such means can the underlying cause be ascertained. He gives histories of cases of retrobulbar and optic neuritis occurring in multiple sclerosis, meningo-encephalitis, tumors of the basal portion of the frontal lobe, toxic amblyopia, lactation and lead poisoning. He believes sinusitis to be an infrequent cause.

W. S. REESE.

RETINAL DETACHMENT AND ITS TREATMENT BY SURGICAL METHODS; A REVIEW OF 425 CASES. C. D. SHAPLAND, Brit. J. Ophth. **18:** 1 (Jan.) 1934.

This paper is a statistical and clinical study. Perhaps its greatest interest lies in a comparison of the results of several operative procedures. This may best be conveyed by the following table, taken from the original article, which shows likewise the chief complications:

Operation	Cure	Improved	Secondary Holes	Vitreous Hemorrhages	Uveitis	Thrombosis Vortex Vein
Cautery puncture						
Holes present.....	29.9	10.3	17.6	7.2	4.5	0.45
"Blind" operation..	0	11.8				
Multiple trephining						
Holes present.....	26.3	14.2	11.4	7.6	2.5	2.5
"Blind" operation..	22.7	18.2				
Diathermy						
Holes present.....	51.7	15.0	6.9	0	2.8	0
"Blind" operation..	25.0	8.3				

W. ZENTMAYER.

PIGMENTATION OF THE PAPILLA. D. MICHAIL, Arch. d'opht. **51:** 129 (March) 1934.

Michail briefly recounts his observations in four cases of pigmentation of the papilla which he has seen over a period of fifteen years. In each one the condition was accompanied by some other clinical compli-

cation. Such cases are quite rare, only forty-six having been found recorded in the literature. The statistics presented confirm the opinions of Ogawa and Fehr that congenital pigmentation is much more frequent than pathologic pigmentation. The appearance, topography, extent and depth of the pigmentation are varied. The vision has been normal in a very limited number of cases, a fact which tends to support the congenital origin. Histologic studies suggest that the condition is more frequent than clinical examinations indicate. The findings of several observers are discussed. Both clinical and histologic studies of this condition indicate that it may be congenital or pathologic. Congenital pigmentation may have its origin from the retina, choroid or glial tissue. The evidence of various observers in support of each is discussed. Pathologic pigmentation most often is hematogenous. Michail correlates his four personal observations with these theories. His first two cases following severe traumas to the head, with the development of a frontal lobe tumor in one, and fracture through the optic canal with consecutive optic atrophy in the second, fall into the pathologic group due to hemorrhage. The third case, associated with zonular cataract, some peripapillary choroidal change and a tigroid fundus, is placed in the congenital group of choroidal origin. The fourth case is placed in the congenital group having origin from the retina. There were accompanying pigmentary changes in the sclera, iris and skin. An extensive bibliography is appended.

S. B. MARLOW.

THE OBLITERATION OF TEARS OF THE RETINA WITH THE GALVANOCAUTERY. P. VEIL and FELGINES, Arch. d'opht. **51:** 158 (March) 1934.

The authors point out the investigations necessary before operation if a lasting cure of detachment of the retina is to be obtained by obliteration of tears. The aim of all the technics proposed is to secure an adhesive choroiditis. The authors are in complete agreement with the statement of Gonin that "the problem is to individualize the cases so that the proper procedure may be applied to each one." They themselves prefer perforating diathermic coagulation because it is less traumatizing and can be controlled ophthalmoscopically. The present paper, however, is for the purpose of showing that galvanocauterization can be successfully employed in several ways to obliterate or wall off retinal tears. They review the various methods which have been proposed. Six cases in which the method of suprachoroidal galvanocauterization according to Rollet and Paufique was used successfully are described. Four of the detachments remained cured at the end of more than a year. One recurred and a second operation was refused. Another recurred twice and was cured by diathermic coagulation. The procedures of Vogt, Fischer, Stargardt and Veil are referred to in some detail. A case of a large detachment with two tears incompletely closed by suprachoroidal galvanocauterization is described. Cure followed diathermic coagulation. Three cases in which the detachments were unsuccessfully treated by diathermic coagulation but cured by galvanocauterization are described.

As a result of these experiences Veil and Felgines point out that galvanocauterization has a place in the treatment of detachment of the

retina. They believe that no one form of technic should be adhered to. They are of the opinion that it is useless to employ diathermic coagulation in those cases in which there is poor response to lying in bed and the wearing of stenopeic glasses. In these galvanocauterization should be used.

S. B. MARLOW.

RETROBULBAR NEURITIS TREATED BY THE OPERATION OF SEGURA HIRSCH. BAILLIART, BALDENWECK and H. TILLÉ, Bull. Soc. d'opht. de Paris, January 1934, p. 22.

In a case of retrobulbar neuritis visual acuity was reduced to 1/50 for a month. Trepanation of the sphenoid sinuses by the septal route restored vision to normal in three days. The general findings, including those of a complete neurologic examination, were negative. The authors believe that the photographs of the fundus show that the recovery was not incidental to the operation, but that the changes were ameliorated as soon as the operation was concluded. Angioscotometry by the method of Evans also confirmed these facts. There was a distinct lowering of the retinal arterial tension following the operation. The authors believe that modern methods of angioscotometry, retinal photography and tonoscopy by the Bailliart method will all aid in enlarging knowledge of this disputable condition.

L. L. MAYER.

THE AQUEOUS HUMOR AND RETINAL DETACHMENT. G. BONNEFON, Compt. rend. Soc. de biol. **115:** 1202, 1934.

According to Bonnefon's conception, circulatory stasis of the aqueous is a primary factor in the type of retinal detachment that accompanies malignant myopia, syphilitic choroiditis and senility. In such cases the pathologic processes have diminished the normal systolic expansion of the choroid by inducing a loss in elasticity of the vessels and involving them in cicatricial adhesions. As evidence of this, the tonometer no longer shows the usual pulsatile oscillations of ocular tension. The stasis of the aqueous that results causes hydration and disorganization of the vitreous. Aqueous seeps through the limiting membrane, and a preretinal collection of fluid ensues. The foci of retinal degeneration present become infiltrated, and finally a subretinal collection forms, with detachment of the retina. The drainage is now deviated toward the choroid, and so continues until checked.

J. E. LEBENSOHN.

RETINAL AND CEREBRAL ANGIOMA. A. NICOLATO, Ann. di ottal. **61:** 736 (Oct.) 1933.

A man of 25 suddenly suffered partial right hemiplegia and paresthesia with dizziness. This was followed three months later by diplopia, left exophthalmos (Hertel's exophthalmometer showed: right eye, 19; left eye, 23) and reduction of vision in the left eye. Vision in the right eye equaled 6/6, and that in the left eye, counting fingers at 50 cm. The left disk was covered by a network of enlarged and tortuous vessels (illustrated). These became much more engorged during pressure on the jugular vein. In one place an arteriovenous anastomosis

could be seen. The visual field of the normal right eye showed a complete nasal hemianopia. Nicolato believes that a hemangioma had involved the left retina, optic nerve, chiasm and internal capsule. He distinguishes the fundus changes in this case from the true hemangioblastomas found in von Hippel's disease which, when combined with similar lesions in the brain, give the picture of Lindau's disease. This puts his case in the group of vascular malformations described by Cushing and Bailey, in which neoplasms do not occur, the damage to the brain and the optic nerve being due to pressure from the enlarged vessels themselves. Apparently no form of surgical treatment was considered advisable.

S. R. GIFFORD.

THE LIPOIDS OF THE RETINA IN VITAMIN A DEFICIENCY. P. PALLIN, Arch. f. Ophth. **131:** 447 (Dec.) 1933.

Sugita produced xerosis conjunctivae, keratomalacia and hemeralopia in white rats by keeping them on a diet lacking only vitamin A. Anatomically the rods were normal, but the pigmented epithelium contained an unusual amount of lipoids and cholesterol esters. This observation Pallin could not confirm in analogous experiments.

P. C. KRONFELD.

EXPERIENCES WITH OPERATIVE TREATMENT OF DETACHMENT OF THE RETINA AND REMARKS ON TRAUMATIC DETACHMENT OF THE RETINA. E. VON HIPPEL, Klin. Monatsbl. f. Augenh. **92:** 145 (Feb.) 1934.

Von Hippel presents a detailed account of his results with operative treatment of detachment of the retina. He operated chiefly after the methods of Gonin and of Weve, choosing the latter's technic of occluding the tear by multiple cauterizations. Arruga's nail was used in operating after the method of Gonin. The method of Lindner and Guist was not employed. Several eyes were cauterized in two and three sittings; in others from two to four cauterizations were done in one sitting.

He considers a tear essential for detachment of the retina in accordance with Leber, who had definitely maintained this view since 1882. In this connection he points out that Leber changed his opinion regarding the forces that produce the tear, and that Leber discussed some details considered of importance at the present time, such as the frequence of tears in the upper temporal portion of the fundus, the occurrence of multiple tears, the peripheral detachment of the retina, and the fact that the margin of the tears is usually turned inward.

Von Hippel considers diathermy far more conservative than the use of Paquelin's cautery, especially when several areas have to be cauterized in one sitting.

Hemorrhages into the vitreous were observed once immediately after the second operation, once after five days and in two cases after a week, and also in three cases in which reattachment was not obtained. His standpoint regarding traumatic detachment of the retina is this: 1. Trauma cannot be held responsible for detachments that occur a year or more after the accident. 2. Concussion of the body or overexertion cannot produce detachment directly. 3. Sport and gymnastic exercises

do not even cause changes in the appearance of the retinal vessels. 4. Sneezing and coughing, as pointed out by Gonin, may produce detachment only in cases in which the retina has been affected to such an extent that the pathologic process responsible for the degeneration of the retina would have sufficed in finally causing it to tear and to detach. Irrelevant injuries, on the other hand, may be followed by detachment, as illustrated by notes on one of his own cases. K. L. STOLL.

Trachoma

BIOMICROSCOPIC OBSERVATIONS IN TRACHOMA VERUM OF THE CORNEA. W. W. TCHIRKOVSKY and L. A. DYMSHITZ, Sovet. vestnik oftal. **4:** 27, 1934.

The authors give a detailed review of the theories and literature of the pathogenesis and biomicroscopy of trachomatous pannus. Nine patients with trachoma of the cornea were examined with the slit lamp; in four of the cases a histologic examination was also made. The ages varied from 9 to 60, and the duration of the disease was from one to twenty-five years. In eight of nine patients no trichiasis, entropion or thickening of the tarsus was observed, i. e., none of the conditions which lead to a mechanical trauma of the cornea. Only in three cases follicles were found in the conjunctiva of the sclera. The technic of the biopsy and the methods of staining are given. The authors describe in detail the slit-lamp and histologic studies of four patients. Their conclusions are as follows:

1. True trachomatous follicles with their typical histologic structures are observed not only in the conjunctiva of the sclera and limbus, but also in the corneal tissue, which gives a typical picture of trachoma verum of the cornea in certain cases.

2. Biomicroscopically the follicles of the cornea present the stereotype form.

3. They are observed in various durations and in various stages of trachoma, but mostly in the regressive period.

4. The authors' clinical and histologic observations in trachoma of the cornea coincide with those of Pasheff.

5. These observations confirm the fact that pannus can appear in a number of cases as a special localization of the trachomatous process in the cornea in its typical follicular form.

Seven excellent photomicrographs are attached.

O. SITCHEVSKA.

Tumors

PRELIMINARY REPORT ON ORBITAL TUMORS. G. HARDY and W. F. HARDY, Am. J. Ophth. **17:** 18 (Jan.) 1934.

Hardy and Hardy discuss orbital tumors mainly from the standpoint of classification. They report three cases more or less in detail and give the following summary: "1. The occurrence of seven orbital tumors in the service of one man in a single hospital group over a period of two years seems disproportionately large. Five of these were confirmed

by operation and of these one was debatable; whether it was a true or pseudonewgrowth is not definitely settled. 2. As a generalization the more anterior the growth the better the prognosis. Those involving the apex, by reason of the close proximity of the important structures subject to pressure, must offer a worse prognosis and a challenge to surgery. 3. Primary tumors of the optic nerve afford a bad prognosis since blindness sets in early and removal is frequently accompanied by the same result. 4. Brilliant results are not to be expected from surgery either with regard to vision or cosmetic appearance. 5. Pseudotumors are difficult of diagnosis and it is not surprising that mistakes in diagnosis and therapy occur. In the case herein recorded the points in favor of tumor were: (a) unilateral exophthalmos, (b) a biopsy with a resultant diagnosis of glioma. Those against glioma were (a) the preservation of fair vision, and (b) the presence of a positive Wassermann reaction. Since the latter is a common finding in the colored race it was natural to discount its importance, especially with a biopsy diagnosis of glioma. Finally, it is fair to assume that blindness due to cicatricial changes, would have eventually occurred even under energetic anti-luetic treatment."

W. S. REESE.

TUBERCLE-LIKE NODULES OF EPISCLERA AND EYELIDS, BILATERAL. W. H. WILMER, Am. J. Ophth. **17**: 99 (Feb.) 1934.

Wilmer cites the case of a woman, aged 56, who showed swelling of both lids of the right eye, especially toward the inner canthus. In the left eye the swelling was confined chiefly to the upper eyelid near the inner canthus. Three lumps could be felt in the right lids, and one in the left upper lid. These did not involve the skin or the eyeball, were freely movable and could be pushed back into the orbit, and were attached to the periosteum. There was a nodule over the right internal rectus which began at its point of insertion and extended back 12 mm. It was about the width of the muscle and showed an elevation of from 2 to 3 mm. It was very hard and not tender. It seemed to be attached to the tendon and sheath of the muscle. There was a similar nodule over the right external rectus, and a long thinner one over the superior rectus. These nodules had the yellowish-red color of an inflamed pinguecula; they did not involve the sclera; the overlying conjunctiva was free, but its vessels showed injection. Vision of both eyes was corrected to 6/6. A piece of the growth over the right internal rectus was removed and examined, also one of the nodules from the lids. Both were typically tuberculous. All the nodules disappeared except that near the internal canthus of the right lower lid. The author discusses the differential diagnosis.

W. S. REESE.

VOLUMINOUS ORBITO-CRANIAL OSTEOMA; CONSECUTIVE CEREBRAL ABSCESS OF NASAL ORIGIN. J. N. ROY, Brit. J. Ophth. **18**: 159 (March) 1934.

The patient was an unmarried woman, aged 24 years, from whom on May 8, 1925, Roy removed an osteoma of the orbit weighing 28 Gm. She had good health until October 1931, when to the accompaniment of a sensation of fulness in the right orbital cavity without pain the eye

slowly proptosed. A growth could be felt by luxating the globe slightly downward. A frontal roentgenogram showed complete opacity of the right orbit. The corresponding frontal sinus was normal. The profile "showed a nippled bony tumor, more voluminous than a large nut, localized forward in the posterior part of the orbit and in the rear of the cranial cavity." There were fever, headache and vomiting. In each eye vision equalled 20/30 (6/9). During the removal of the osteoma, which had its origin in the ethmoid sinus, 20 cc. of pus flowed from the frontal lobe. The patient died twenty-eight hours after the operation from leptomeningitis.

The postmortem examination showed an absence of the roof of the orbit. The growth had developed on the old cicatricial tissue of the orbit as a fibroma which in the process of time had progressively undergone ossification. It is assumed that the microbes coming from the nose had proceeded to the brain by the cortical part of the osteoma. The article is illustrated.

W. ZENTMAYER.

A TUMOR OF THE BASE OF THE IRIS NOT PREVIOUSLY DESCRIBED. J. MAWAS, Bull. Soc. d'opht. de Paris, February 1934, p. 40.

The patient complained of visual loss, some pain and redness. Direct examination demonstrated a new growth in the anterior chamber and hypertension of the globe. The previous history was unimportant except that three years before examination the patient was hit in the eye by a piece of coal. There was only a slight redness of the eye at that time. With the slit lamp the findings were: a normal cornea, a clear anterior chamber, no precipitates on Descemet's membrane, an apparently normal iris with regular border, a transparent lens and vitreous and a tumor situated to the external side between 1 and 4 o'clock, protruding into the anterior chamber and touching the posterior corneal surface. This growth was oval with its greatest axis horizontal and pointing toward the pupil. It did not adhere to the iris completely but seemed to lie on it. The pupillary reactions were conserved. The tumor was rich in blood vessels but without pigmentation. The entire body of the tumor was translucent except for the vessels. After the glaucoma had been treated with pilocarpine for several days the tumor was removed by a broad iridectomy. Tension was lowered but rose after a month to 70 mm. A Lagrange operation was done. Histologic examination revealed no traces of an inflammatory reaction, no pigment, no caseation or necrosis and no lymphoid or plasma reactions. Neither spirochetes nor tubercle bacilli were present. The author wishes to call this type of tumor a nevocarcinoma.

L. L. MAYER.

THE HISTOGENESIS OF TUMORS OF THE RETINA. E. REDSLOB, Bull. Soc. d'opht. de Paris, March 1934, p. 123.

Redslob believes that the polymorphism displayed by tumors of the retina should be considered as groups of cells of the central nervous system in certain stages of embryogenesis. All tumors of the retina are composed of identical elements. The characteristics which make the tumors different are simply the various arrangements of these cells.

Wintersteiner and Mawas consider the small round cells found in such abundance in neuro-epithelioma as retinal embryonic cells, the rosettes as different types of young visual cells. Redslob believes these to be very young cells of the central nervous system in a certain stage of development. He confirms this a priori, as it is known that the retina in its origin is simply an evagination of the ectoderm of the brain.

Tumors of the retina and the neurospongiomas have many points in common. The formation of ependymal cells represents one of the first stages in the differentiation of the neurospongiomas. It is to Hortega and his method of study that one must look for further classification. The term "retinocytoma" is improper, for the cells of the retina proper do not contribute in any way to making up the tumors of the retina. References and a discussion are given.

L. L. MAYER.

HAMARTOMA OF THE LACRIMAL CARUNCLE. S. SGROSSO, Arch. di ottal. **41**: 90 (Jan.-Feb.) 1934.

An infant was seen with a mass the size of a large pea in the inner angle of the eye. The globe was microphthalmic with a cataractous lens. The mass arising from the caruncle was adherent to the sclera. It was removed without injuring the globe, and the inner angle was reconstructed with the remaining skin and conjunctiva. The tumor measured 17 by 12 mm. It contained fatty tissue with some glandular tissue. Since the elements present showed the characteristics of normal tissue the tumor is classed as a hamartoma (Albrecht) and not as a blastoma. Its presence in fetal life had apparently interfered with the normal development of the globe.

S. R. GIFFORD.

Uvea

BLOOD CULTURE STUDIES IN IRITIS. E. F. TRAUT, Am. J. Ophth. **17**: 106 (Feb.) 1934.

Traut gives the following summary: "Using a modified technic developed by Clawson for studying the streptococci in the peripheral blood, five patients with acute iritis yielded pleomorphic streptococci. These organisms resembled morphologically and culturally those isolated from blood of arthritic patients.

"Two of the five patients with iritis had had frank arthritis. Three of the patients had had recurrent attacks of iritis. In two the disease followed acute upper respiratory disease. Acute upper respiratory disease had preceded the iritis in a patient with lues. Whether the lues was a factor in producing the iritis is a moot point not easily settled even by a favorable outcome following antiluetic medication."

W. S. REESE.

Vision

THE TESTING OF VISUAL ACUITY. C. E. FERREE AND G. RAND, Am. J. Ophth. **17**: 29 (Jan.) 1934.

Ferree and Rand remark that there are three important factors in the visibility of the image on the retina: its clearness, its subjective

or sensational difference from the background, and its size. There are two ways of decreasing the sensational difference. One is by decreasing the difference in the coefficients of reflection as by using gray test objects on white backgrounds or black letters on a gray background. Another way is to use a fairly high difference in coefficient of reflection, as is the case of the customary black on white, and decrease the intensity of illumination. These two procedures are discussed together with size of the pupil in connection with the latter. The methods used are described and the results tabulated. The authors conclude that: 1. Much smaller errors of refraction can be detected when the coefficient of reflection is small than when it is large. 2. When the test object is black and the background white the amount of refractive error that may remain in an eye with good powers of vision, when the refractionist does not attempt to correct to better than 6/6, is rather astonishingly large. 3. When the test object is black and the background gray the amount of refractive error that may escape detection by use of the 6/6 line on the chart is less.

W. S. REESE.

Therapeutics

THE TREATMENT OF FOLLICULAR TUBERCULOSIS OF THE EYE BY MEANS OF A METHYLIC ANTIGEN. BEAUVIEUX, Arch. d'opht **50:** 540 (Aug.) 1933.

Beauvieux presents the point of view that ocular tuberculosis is comparable to surgical tuberculosis. It corresponds to the tertiary stage of the disease, the first stage being that of inoculation, and the second that of bacillemia and the allergic reactions. Although this conception of the disease was first suggested by Ranke it had been entertained by many for some time and offers a sounder basis for treatment with tuberculin. Beauvieux makes no attempt to review the literature of tuberculin therapy but was impressed with the use of the vaccine of Nègre and Boquet in the treatment of a follicular tuberculosis of the eye, the rheumatic type of Poncet. He reports two cases in which the use of this vaccine was followed by surprising improvement. Commenting on the two observations he points out the possibility in some instances of curing tuberculosis of the uveal tract. In his opinion the vaccine of Nègre and Boquet is preferable to all others because of its harmlessness and safety in inexpert hands and because of its well regulated dosage. The radical cures obtained in the two cases reported should be of special interest. The results published by Rousseau and Spillman strongly support his contention. He believes, however, that further trial is necessary before the value of this treatment can be finally estimated.

S. B. MARLOW.

BLINDNESS DUE TO HEMORRHAGE CURED BY ACETYLCHOLINE. E. HARTMANN and J. PARFONRY, Bull. Soc. d'opht. de Paris, February 1934, p. 56.

A patient had a gastric hemorrhage due to an ulcer. Twenty-four hours after the hemorrhage marked loss of vision developed. Transfusion failed to restore vision. There was a loss of color in the optic disks with peripapillar hemorrhages. In one month's time complete

blindness ensued. An injection of 0.1 Gm. of acetylcholine was given every two days for twenty days. After the third injection vision began to return, being 5/35 for the right eye and 5/20 for the left. The temporal fields of vision became almost normal, but there was complete loss of the nasal fields. Vision was recovered to 5/10 in each eye. The authors stress the following points: (1) the efficacy of acetylcholine even in long-standing cases of visual loss; (2) the use of a vasodilator in vascular lesions of the eye; (3) the loss of the nasal fields while the temporal portions became approximately normal; (4) the loss of nasal fields as due to vascular stasis, which is more difficult to treat. A discussion and bibliography are included.

L. L. MAYER.

AUTOGENOUS VACCINE AND CHRONIC SECRETION OF ENUCLEATION CAVITIES. HUDELO AND BRUNEAU, Bull. Soc. d'ôpht. de Paris, March 1934, p. 95.

Cavities having chronic secretions following enucleation or evisceration were treated by means of vaccines made from the secretions.

The patients were divided into three categories: (1) those in whom a vaccine was used before the patient was wearing a prosthesis; (2) those in whom a vaccine was used who had had an artificial implantation for many years, the cavities being normal but the glass eye not comfortable; (3) those in whom a vaccine was used in whose cavities a chronic secretion, an abscess or a phlegmon had occurred and whose prosthesis was carried without comfort.

Twenty case reports reveal the types of patients treated. The entire technic of the bacteriologic investigation is outlined. Charts of the bacteria found in the secretion and in the cultures and those used for the vaccine are shown.

Injections of the vaccine were made subcutaneously beginning with 0.25 cc. and increasing by 0.25 cc. at intervals of every other day, up to 1 cc. From ten to twelve injections were given. In the first group without prosthesis two patients treated were cured. In the second group there were nine patients. Six were cured, two were markedly benefited and one was not helped. Of those with abnormal cavities, four were cured and in two the condition was checked. In two cases, dacryocystitis was completely arrested. Retreatment of uncured patients resulted in some benefit.

L. L. MAYER.

Toxic Amblyopia

DISTURBANCES OF VISION AFTER THE INJECTION OF ACETYLARSAN. TISSOT-DAQUETTE, Arch. d'opht. **51**: 17 (Jan.) 1934.

To the fifteen cases already reported the author has added four unpublished cases in her thesis. In this paper she discusses briefly the conclusions which can be drawn from the observations made. Acetylarsan belongs to the group of pentavalent arsenicals. Like the others it is able to produce lesions in the visual nerve apparatus. These lesions are bilateral and of the same degree in both eyes, and in the majority of instances terminate in total or partial atrophy of both optic nerves.

The subjective and functional symptoms vary according to the patient, but the objective findings are always the same. The involvement of the visual fibers generally comes on some hours after an injection. It begins sometimes with a sudden complete amaurosis, sometimes with a varying loss of vision due either to a concentric contraction of the field of vision with or without lowered central acuity, or to a simultaneous peripheral and central diminution, or very rarely to a central scotoma. Sometimes the ocular involvement consists simply of very mild subjective disturbances.

In the very early stages either no changes can be made out on ophthalmoscopic examination or, at the most, occasionally some constriction of the retinal vessels. After a short time in the majority of cases there appears a pallor of the disks which goes on to optic atrophy. These visual symptoms sometimes improve but more often are resistant to all treatment. The patients become blind or have very defective vision. These accidents consequently are very serious but on the other hand are very rare. Acetylarsan seems to be toxic to the visual apparatus only under certain conditions: (1) when improperly administered, (2) when there are predisposing factors, such as a previous lesion of the optic nerve or deeper membranes, a former involvement of the central nervous system, especially of the meninges, renal and hepatic insufficiency, alcoholism, general debility and senility. It therefore appears that visual accidents are avoidable if the contraindications are heeded and correct administration is used.

S. B. MARLOW.

Society Transactions

ROYAL SOCIETY OF MEDICINE, LONDON, SECTION OF OPHTHALMOLOGY

Oct. 12, 1934

MR. RANSOM PICKARD, C.B., M.S., *Presiding*

RETINAL DETACHMENT IN AN APHAKIC EYE CURED BY ŠAFÁŘ'S METHOD. MR. T. C. SUMMERS.

The patient, a woman, aged 62, was first seen on July 2, last, with the complaint that vision in the left eye had been suddenly lost two weeks before. A large billowing detachment was seen, affecting more than half of the upper part of the retina. The lens had been removed a year prior to this. A small round hole was found in the 10 o'clock position, 4 disk diameters from the ora serrata. Vision was less than 6/60.

In the right eye there was only perception of light, this condition having followed an operation for cataract two years before.

Forty-six punctures were made with a current of 70 ma., and an uninterrupted recovery ensued. Ten days ago vision was 6/9 partly. The whole retina is still in situ, and the site of the operation is indicated by a large mass of old choroiditis. There has been left, however, a defect of vision for a fine object in that part of the field which was originally the best.

TUMOR OF THE IRIS. MR. A. F. MACCALLAN.

This patient came to the hospital last month asking to be examined for glasses. She was short-sighted, having 7 diopters of myopia in one eye and 10 diopters in the other. A small tumor was found in the iris of the left eye.

DISCUSSION

MR. GRAY CLEGG: I think this is a congenital condition; I have seen a case with a similar appearance. I should watch it for several months before deciding on operative treatment.

BILATERAL PROPTOSIS—A SYNDROME RELATED TO THE SPHENOID FISSURE. MR. EUGENE WOLFF and MR. E. D. MACKWORTH.

Following a cold, this woman, aged 50, had a severe headache with a loud throbbing noise in the head, accompanied by vomiting. Her temperature was 100 F.; her pulse and respiration were normal. The left eye was markedly proptosed; it did not pulsate, and the sight was not affected. The movements of the eye were limited in all directions. The left pupil was smaller than the right. There was a well marked deflection of the septum of the nose to the left, but there was no pus in the nose. X-ray pictures revealed nothing abnormal in the sinuses. A

loud blowing systolic murmur was audible all round the left orbit. In a fortnight the condition had largely subsided, but the murmur remained, as also did the limitation of outward movements. After eight weeks the proptosis was less, the injection of the conjunctiva not so marked, and the movements freer. X-ray pictures did not show any abnormality of the sphenoid fissures. The pulse rate was consistently 80, and there was no enlargement of the thyroid gland.

DISCUSSION

MR. D. V. GIRI: A mild ethmoiditis might have set up orbital cellulitis; the latter may have cleared up by the time the roentgenogram was taken.

MR. TAIT: There are two points against its being ethmoiditis: the marked proptosis and the systolic bruit.

MR. F. A. WILLIAMSON-NOBLE: I had a similar case a good many years ago, in which the eye was removed and the orbit exenterated. The proptosis had begun after an attack of influenza. When I examined the eye pathologically, all that I could find was a mass of lymphocytes. Perhaps if the surgeon in charge had exercised patience and watched the eye for a time it could have been saved. There was no record of any bruit.

TRAUMATIC RETINITIS PROLIFERANS. MR. A. RUGG-GUNN.

A girl, aged 10 years, fell against the corner of a chair when she was 10 months old, injuring the left eye, which became proptosed and divergent. Vision is now: right eye, 6/5; left, 6/60. The pupils react. There is upward deviation of the left eye, but its movements are unimpaired. There is a bluish-white mass occupying the central portion of the vitreous; it ends in the anterior portion of the vitreous in a rosette of pointed processes from which fibrils pass to the back of the lens and perhaps to the anterior part of the fundus. The slit lamp shows a series of slack delicate attachments to the back of the lens. There is degenerative choroiditis, without pigment, in the fundus. There are no vessels visible in the new formation.

DISCUSSION

MR. D. V. GIRI: I saw a boy who received a dart in the eye. I was consulted within an hour after the accident. The dart was from 3 to 4 mm. in diameter and was plucked out by the boy himself. I could not find that the sclera was perforated. I admitted him to the hospital and treated him with injections of milk, and for a long time I kept him on small doses of thyroid and iodine. There was hemorrhage into the vitreous, and from the spot of perforation thin sheets like a fan spread into the vitreous. At one time vision was below 6/60. Vision is now 6/5.

THE CAUSATION OF HERPES OPHTHALMICUS. MR. RANSOM PICKARD.

The main clinical fact established in late years is the relationship of herpes zoster to varicella. Either disease may cause the other; less frequently, they may coexist in the same person. That these two phases are unstable is shown by the moderate frequency with which they

are transmuted in the passage of the disease from one person to another; but the varicella phase is the more infectious as such. Herpes corneae has two phases, the dermatotropic and the neurotropic, and there is some interchangeability between the two strains of herpes corneae, though the encephalitic variety becomes more specialized and less transmutable than the dermatotropic.

Head and Campbell showed that the main seat of inflammation is in the posterior root ganglion, and that the inflammation spreads up and down the nerve to a slight extent. The degeneration sometimes spreads up into the posterior column of the cord and down the sensory nerve and its branches to their entry into the cutis. In two cases, no lesions of the ganglions and nerves were found eight months after the attack.

The eye attacked with herpes ophthalmicus is severely affected with iritis and hypopyon. In cases in which the iris was excised, it was found to be necrosed in the lower part, and the adjoining portions of the ciliary body and sclerotic body were involved. Retinitis and choroiditis have not been observed with the herpes ophthalmicus. In one case, in the uvea near to the herpetic area, in front of the ora serrata, was a small nodule. The retina was unaffected except by a few infiltrating mononuclear cells. The optic papilla was slightly edematous and contained a few round cells. The optic nerve showed destruction and fibrillation of the medullary sheaths of the nerve fibers. The ciliary nerves showed marked changes: They were surrounded by a sheath of mononuclear cells, the endoneurium also showing many of these. But the ciliary vessels were not affected. The suggestion that the cutaneous eruption in herpes zoster is due to an alteration in the trophic impulses from the affected posterior root ganglion or spinal cord is not, in the author's opinion, adequate in explaining the groups of vesicles in the skin or the manifestations in and about the eye. Levaditi expressed the belief that there is a normal *"tonus immuno-trophique"* in each spinal segment which prevents infection, and that lowering of this enables the organism to establish itself in the segment or in that part of the body supplied by it. Head and Campbell suggested that the production of the rash is due to an intense irritation in the cells in the ganglion which normally subserves the function of pain, particularly that form of pain produced by afferent visceral impulses. If the rash were due to increased activity in the posterior root ganglion, causing an increase in the normal trophic impulses, it would be expected to produce a more uniform effect on the skin than it does, for the lesions in the ganglion are usually diffuse, not confined to limited areas. In a case of herpes sufficiently severe to cause vesicles to appear in all parts of the area of skin corresponding to the ganglion affected, the skin between the individual vesicles is often found to be not even red. So there is selectivity. Within a group the individual vesicles tend always to coalesce. It is a fair conclusion that the vesicles are caused by the exciting organism itself, acting from a distance by nerve impulses, by transmission of the toxin along the nerves or by its own actual passage to the skin. A toxin may be excluded.

It has been proved that in the herpes zoster phase the organism has a special affinity for the posterior root ganglions. Let it be assumed that the organism normally spreads thence along the sensory nerves to

the tissues supplied by them and that on reaching the fibrous tissue covered by cells of ectodermal origin it reverts to the varicella phase; let it also be assumed that in some cases the organism may spread locally from the posterior ganglion, thus reaching adjoining structures. The usual sequence is that pain occurs three or four days before the rash appears. The interval represents the growth of the organism along the nerves. More difficult to explain is the late occurrence of keratitis, iritis and cyclitis.

In regard to lesions other than those of the skin and eyeball, there is a rare complication of herpes ophthalmicus, a lesion of the optic nerve resulting in complete and permanent blindness. There is no anatomic or developmental connection between the optic nerve and the gasserian ganglion, but they are only from 2 to 3 mm. apart. Such a case is most simply explained by a local spread to the optic nerve. Similar reasoning applies to paralyses or pareses of the third, fourth and sixth nerves when the fact is invoked that the gasserian ganglion is the posterior root ganglion for these nerves.

Book Reviews

Al Morchid Fi'l Kohhl (The Ophthalmologic Guide). By Mohammad Al-Ghafiqi. Abridged French translation from the Arabic edition by Max Meyerhof, M.D., Ph.D. Pp. 225, with 14 illustrations. Barcelona: Laboratoroires du Nord de l'Espagne Directeur, Masnou, 1933.

Again Dr. Max Meyerhof, the well known ophthalmologist and tranlator of Arabic literature into English (ARCH. OPHTH. **2**: 114 [July] 1929) and French, has rendered valuable service in the translation of this treatise.

The translation of the Arabic edition was made for the recent ophthalmologic congress at Madrid. The text was written in the twelfth century. A brief analysis can be found in Leclerc's "History of Arabic Medicine" (Paris, 1876). The book begins with the famous oath of Hippocrates and continues with a prolonged discussion of the dignity and eminence of medical art. Mohammad Al-Ghafiqi discusses questions pertaining to medicine, anatomy, etc., in a typically scholarly manner.

The diseases of the eyelids are classified into thirty-seven different varieties, including trachoma. The descriptions are clear, distinct and lucid and almost meet the modern conception of the conditions of the eyelids. The treatment is good and in many ways superior to that of works of the previous centuries. Minute details are given regarding the general care of the health. Venesection, hydrotherapy and indifferent purgatives form the basis of this general treatment.

For local treatment the extensive use of zinc, silver and copper is recommended.

Following descriptions of phlegmon, inflammation and epiphora of the lacrimal sac, thirteen different conditions involving both the conjunctiva and the cornea are described. In regard to these, Mohammad Al-Ghafiqi again lays much stress on the general treatment of the patient. Not only does he favor local treatment, but the frequent use of surgical measures is recommended.

His conception of cataract is that it is a condensation between the cornea and iris; he classifies it into operative and inoperative forms, but demands that the light sense be tested and found present before operating.

A detailed method for couching the lens is described.

Meyerhof terminates his work by stating that ophthalmology did not advance much further from the thirteenth to the eighteenth century than its status in the twelfth century.

Following the translation, Meyerhof gives an excellent résumé of ocular treatment in antiquity and in the Middle Ages.

He also states that drops instilled into the conjunctival sac have been used only in the past one hundred and fifty years; previously, a paste was made of the desired drug, and this was placed in the conjunctival sac.

ISADORE GOLDSTEIN.

Handbuch der biologischen Arbeitsmethoden. Edited by Prof. E. Abderhalden. Div. 5, part 6: **Lichtsinn und Auge. Methoden zur Untersuchung der vegetativen Funktionen des Auges und der Leistungen seiner Anhangsorgane.** By K. Velhagen jun. Halle a. d. S. **Die Methoden der Ophthalmographie (Augenhintergrundphotographie).** By J. W. Nordenson, Stockholm. Price, 6 marks. Pp. 100, with 56 illustrations. Berlin: Urban & Schwarzenberg, 1934.

Dr. K. Velhagen has written a short description of the experimental surgery practiced on laboratory animals in the study of autonomic functions of the eye. There are taken up in turn the instruments, narcosis and exposure of the eyeball; then follows a description of the operations on the external muscles, the extra-ocular vessels and nerves, the glands of the orbit, the lacrimal canal and the eyeball. In the chapter on physiologic experiments the following are described: experiments on the surviving eye, when attached to the head, and on the surviving inner ocular muscles, the pupillary reaction, the intra-ocular circulation, the pulling power of the ocular muscles, lacrimal function and corneal pulsation.

This is an excellent exposition of the various procedures which are being used in the laboratory and will be of great assistance to those who are working on these problems, particularly if they are not fully conversant with the technic of the physiologic laboratory.

Professor Nordenson, one of the pioneers in fundus photography, describes the methods of ophthalmography (fundus photography). The advantages of fundus pictures are now so well recognized that a thorough description of the apparatus and directions for its use are most timely. Color photography, Nordenson states, has its difficulties in the limited time of exposure. The best colored pictures have been obtained by the "subtractive" method, in which certain parts of the spectrum are subtracted from the white light. Three pictures each in the basic colors, blue, yellow and red, are first taken. The plates are then superimposed so that a colored picture results. This procedure has been worked out by the Amatcolor Company for color photography in Hamburg.

ARNOLD KNAPP.

CORRECTIONS

In the article by Dr. Harry Eggers, "Power of Ophthalmic Lenses," in the November issue (**12**:753, 1934) a wrong line was inserted in the third paragraph under the center head "Distance of Lens from the Eye," on page 760. The fourth line in this paragraph should read: "would act as a minus 14.3 diopter sphere (because $C = 0.001 \times 3 \times$."

In the November issue it was inadvertently stated that Dr. Vito La Rocca was not connected with the Brooklyn Eye and Ear Hospital. The statement should have been that the research work he reported in the October issue, entitled "Experimental Albuminuria in Rabbits," was completed under the auspices of the pathology department of the Brooklyn Eye and Ear Hospital, not under the auspices of the research department of the Brooklyn Eye and Ear Hospital.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Prof. F. de Lapersonne, 217 Faubourg St. Honoré, Paris.
Secretary-General pro tem: Dr. A. Churchill, 66 Boulevard St. Michel, Paris.

International Ophthalmologic Congress

Secretary: Dr. Francisco Poyales, Olozaga 3, Madrid.
Place: Cairo. Time: 1937.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. A. J. Ballantyne, 11, Sandyford Pl., Glasgow, C 3.
Secretary: Dr. J. Ringland Anderson, 108 Collins St., Melbourne.
Place: Melbourne. Time: Sept. 11-13, 1935.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Ophthalmological Society of the United Kingdom

President: Mr. A. H. H. Sinclair, 6 Charlotte Square, Edinburgh.
Secretary: Mr. Humphrey Neame, 56 Portland Place, London, W. I.

Oxford Ophthalmological Congress

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Place: Oxford. Time: July 4-6, 1935.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. A. C. Hudson, 50, Queen Anne St., W. 1, London.
Secretary: Dr. J. D. Magor Carbell, 27, Weymouth St., W. 1, London.

Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.

Society of Swedish Ophthalmologists

President: Prof. F. Berg, Uppsala, Sweden.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

German Ophthalmological Society

Secretary: Prof. A. Wagenmann, Heidelberg.

NATIONAL

American Medical Association, Scientific Assembly, Section on Ophthalmology

Chairman: Dr. Arthur J. Bedell, 344 State St., Albany, N. Y.
Secretary: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Place: Atlantic City, N. J. Time: June 10-14, 1935.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

American Academy of Ophthalmology and Otolaryngology

President: Dr. J. M. Wheeler, 635 W. 165th St., New York.
President-Elect: Dr. Wells P. Eagleton, 15 Lombardy St., Newark, N. J.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Cincinnati. Time: 1935.

American Ophthalmological Society

President: Dr. Walter B. Lancaster, Boston.
Secretary-Treasurer: Dr. J. Milton Griscom, 2213 Walnut St., Philadelphia.
Place: Hot Springs, Va. Time: June 5-7, 1935.

National Society for the Prevention of Blindness

President: Mr. William Fellows Morgan.
Managing Director: Miss Regina E. Schneider, 50 W. 50th St., New York.

SECTIONAL

New England Ophthalmological Society

President: Dr. Hugo B. C. Riemer, 128 Newbury St., Boston.
Secretary-Treasurer: Dr. Benjamin Sachs, 483 Beacon St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. Frank B. Kistner, Mayer Bldg., Portland, Ore.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: Portland, Ore. Time: May 20-23, 1935.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. Francis A. Brugman, 509 Olive St., Seattle.
Secretary-Treasurer: Dr. Noble Dick, 1115 Terry Ave., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

Rock River Valley Eye, Ear, Nose and Throat Society

President: Dr. Harry Shearer, 405 E. Grand Ave., Beloit, Wis.
Secretary-Treasurer: Dr. W. S. Beyer, 1101 Talcott Bldg., Rockford, Ill.
Place: Rockford, Ill., Janesville or Beloit, Wis. Time: Third Tuesday of each month.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. Charles H. Baker, 805 Washington Ave., Bay City, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: First Friday of each month, except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. J. J. Hompes, 1307 N St., Lincoln, Nebr.
Secretary-Treasurer: Dr. J. C. Decker, Francis Bldg., Sioux City, Iowa.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. William A. Wagner, 914 American Bank Bldg., New Orleans.
Secretary: Dr. O. M. Marchman, Medical Arts Bldg., Dallas, Texas.
Place: St. Louis. Time: November 1935.

Southwestern Michigan Triological Society

President: Dr. F. B. Fralich, 203 S. Main St., Ann Arbor, Mich.
Secretary-Treasurer: Dr. A. S. Barr, 201 S. Main St., Ann Arbor, Mich.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Claude W. McKee, 229 S. Main St., Greensburg.
Secretary-Treasurer: Dr. C. W. Beals, 41 N. Brady St., DuBois.
Place: Indiana. Time: October 1935.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Edna M. Reynolds, 227, 16th St., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Arthur M. Yudkin, 257 Church St., New Haven.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.
Time: May, November.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. Herschel C. Crawford, 26 Lindon Ave., N. E., Atlanta.
Secretary-Treasurer: Dr. J. A. Smith, 700 Spring St., Macon.
Place: Atlanta. Time: May 7-10, 1935.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Ora G. Brubaker, North Manchester.
Secretary: Dr. Frederick V. Overman, 705 Hume-Mansure Bldg., Indianapolis.
Place: Indianapolis. Time: April 10, 1935.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. F. W. Dean, 532, 1st Ave., Council Bluffs.
Secretary-Treasurer: Dr. O. L. Thorburn, 213½ Main St., Ames.
Place: Council Bluffs. Time: 1935.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. John W. Orr, 503 S. Saginaw St., Flint.
Secretary: Dr. D. R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. James A. Reynolds, 78 S. 9th St., Minneapolis.
Secretary-Treasurer: Dr. Walter E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. Lawrence G. Dunlap, 101 Main St., Anaconda.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY, OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. James A. Fisher, 501 Grand Ave., Asbury Park.
Secretary: Dr. William K. Campbell, 96 Third Ave., Long Branch.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. Webb W. Weeks, 20 E. 53rd St., New York.
Secretary: Dr. John F. Fairbain, 925 Delaware Ave., Buffalo.
Place: Albany. Time: May 13-15, 1935.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. W. R. Winn, 702 First Ave., S., Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 516 Fourth St., Valley City.
Place: Minot. Time: June, 1935.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. Nathan Bolotow, 108 Waterman St., Providence.
Secretary and Treasurer: Dr. Gordon J. McCurdy, 122 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Walter Bristow, Medical Bldg., Columbia.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Eugene Orr, 706 Church St., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 248 Madison Ave., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. T. E. Fuller, 100 W. Broad St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. William Stookey, 54 E. South Temple St., Salt Lake City.
Secretary-Treasurer: Dr. T. F. Welsh, 711 Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Beverley R. Kennon, 142 W. York St., Norfolk.
Secretary-Treasurer: Dr. George G. Hankins, Medical Arts Bldg., Newport News.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. W. F. Beckner, 1050 Fifth Ave., Huntington.
Secretary: Dr. F. O. Marple, First Huntington National Bank Bldg., Huntington.
Place: Wheeling. Time: May 1935.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Alfred Stahl, 55 Lincoln Park, Newark, N. J.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Herschel Crawford, 478 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. Stacy C. Howell, 384 Peachtree St., N. E., Atlanta, Ga.
Place: Academy of Medicine, 13 Queen's Park. Time: 8 p. m., last Thursday of each month.

BALTIMORE MEDICAL SOCIETY. SECTION ON OPHTHALMOLOGY

Chairman: Dr. Lloyd B. Whitham, Mt. Vernon Pl. and Charles St., Baltimore.
Secretary: Dr. Mary L. Small, 18 W. Read St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. John H. Bailey, 855 St. Marks Ave., Brooklyn.
Secretary-Treasurer: Dr. George Freiman, 703 Grand St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. H. H. Glosser, 448 Franklin St., Buffalo.
Secretary-Treasurer: Dr. I. J. Koenig, 40 North St., Buffalo.
Time: Second Thursday of each month.

Chicago Ophthalmological Society

President: Dr. E. V. L. Brown, 122 S. Michigan Ave., Chicago.
Secretary-Treasurer: Dr. Thomas D. Allen, 122 S. Michigan Ave., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. A. L. Stotter, 1148 Euclid Ave., Cleveland.
Secretary: Dr. James T. Collins, 1105 Medical Arts Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. W. P. Chamberlain, 7405 Detroit Ave., Cleveland.
Secretary: Dr. A. D. Ruedemann, Cleveland Clinic, Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. John Milton Griscom, 2213 Walnut St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. W. C. Davis, 327 E. State St., Columbus, Ohio.
Secretary-Treasurer:. Dr. H. M. Sage, 9 Buttles Ave., Columbus, Ohio.
Place: Athletic Club. Time: First Monday of each month.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. W. Mood Knowles, 1719 Pacific Ave., Dallas, Texas.
Secretary: Dr. L. H. Quinn, 4015 Live Oak St., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. C. C. Walker, 410 6th Ave., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month, September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Raymond S. Goux, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. Arthur F Holding, 142 Washington Ave, Albany.
Secretary-Treasurer: Dr. W. C. Mott, 214 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. T. L. Goodman, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

GRAND RAPIDS EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. John H. McRae, 26 Sheldon Ave., S. E., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 307 Metz Bldg., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

HOUSTON ACADEMY OF MEDICINE, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. R. M. Hargrove, 1202 Esperson Bldg., Houston, Texas.
Secretary: Dr. J. C. Dickson, 1617 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. C. H. McCaskey, 404 Continental Bank Bldg., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Raymond E. Teall, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. A. W. McAlester, 2003 Bryant Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. Russell T. Uhls, 110 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. Kenneth C. Brandenburg, 707 Security Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. F. H. Brandt, 1136 W. Sixth St., Los Angeles.
Secretary-Treasurer: Dr. Pierre Viole, 1930 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

LOUISVILLE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Charles Dwight Townes, 332 W. Broadway, Louisville, Ky.
Secretary-Treasurer: Dr. J. S. Bumgardner, 1103 Heyburn Bldg., Louisville, Ky.
Place: French Village. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

Chairman: Dr. R. S. Pendexter, 900, 17th St., N. W., Washington.
Secretary: Dr. Lyman B. Tibbets, 1801 Eye St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, 130 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. J. J. Brook, 231 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. Herbert G. Schmidt, 238 W. Wisconsin Ave., Milwaukee
Place: University Club. Time: 6:30 p. m., fourth Tuesday of each month.

Montreal Ophthalmological Society

President: Dr. J. A. MacMillan, 1410 Stanley St., Montreal, Canada.
Secretary: Dr. A. L. Guertin, 410 Sherbrooke St., West, Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Wesley Wilkerson, Bennie Dillon Bldg., Nashville, Tenn.
Secretary-Treasurer: Dr. Hale Cullom, Bennie Dillon Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to June.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Webb William Weeks, 20 E. 53d St., New York.
Secretary: Dr. Guernsey Frey, 121 E. 60th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. John H. Judd, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. Robert H. Rasgorshek, 425 Aquila Court, Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Secretary: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Edward B. Heckel, 500 Penn Ave., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Pittsburgh Slit-Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. F. H. Lee, 501 E. Franklin St., Richmond, Va.
Secretary: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. Frank Barber, 75 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Laurence Macey, 221 Alexander St., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. E. C. Spitze, 234 Collinsville Ave., East St. Louis, Ill.
Secretary: Dr. Carl T. Eber, 1006 Carleton Bldg., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Robert E. Parrish, 712 Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. Frank Steed, Nix Professional Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Frank H. Rodin, 490 Post St., San Francisco.
Secretary: Dr. Louis F. Morrison, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Fourth Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Secretary-Treasurer: Dr. Leon F. Gray, 1214 Slattery Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 8:15 p. m., fourth Tuesday of every month except June, July and December.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Marc Anthony, 422 Riverside Ave., Spokane, Wash.
Secretary: Dr. Raymond A. Lower, 1455 Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. D. F. Gillette, 109 S. Warren St., Syracuse, N. Y.
Secretary-Treasurer: Dr. W. J. Werfelman Jr., 725 State Tower Bldg., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Clarence E. Hill, 160 Bloor St., West, Toronto.
Secretary: Dr. A. Lloyd Morgan, 86 Bloor St., West, Toronto.
Time: First Monday, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. William Thornwall Davis, 927 Farragut Sq., Washington, D. C.
Secretary-Treasurer: Dr. James N. Greear Jr., 1740 M St., N. W., Washington, D. C.
Place: Episcopal Eye and Ear Hospital. Time: 8 p. m., first Thursday in November, January, March and May.